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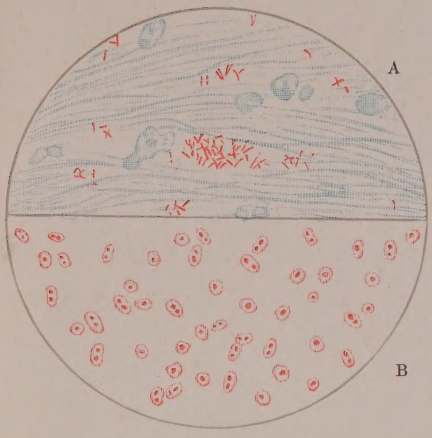
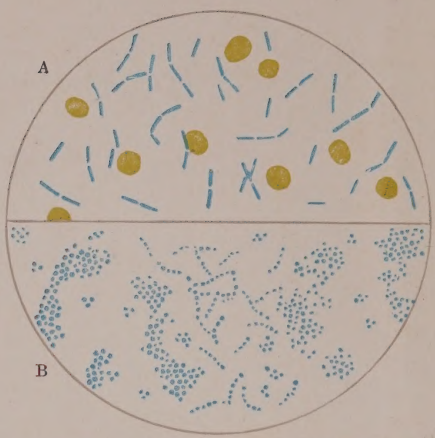
PLATE I.

FIG 1.



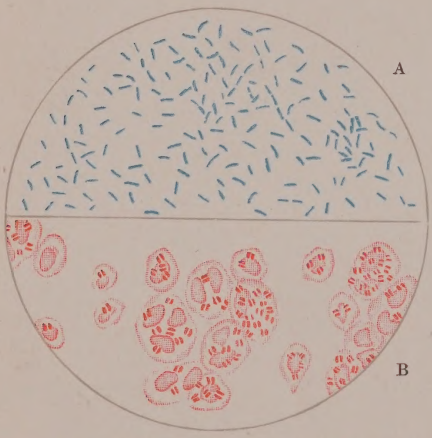
A. Tubercle Bacilli. B. Pneumococcus.

FIG. 2.



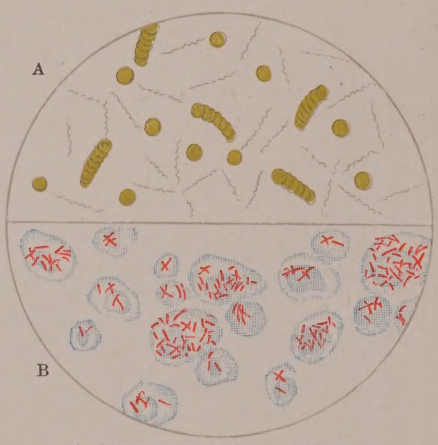
A. Anthrax. B. Streptococcus and Staphylococcus.

FIG. 3.



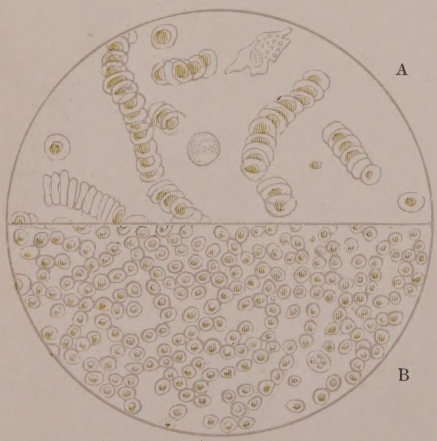
A. Comma Bacillus. B. Gonococcus.

FIG. 4.



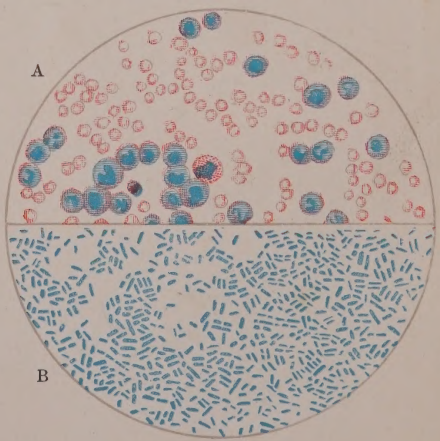
A. Recurrent Spirilla. B. Leprosy.

FIG. 5.



A. Normal Blood. B. Normal Blood.

FIG. 6.



A. Leukæmia. B. Eberth's Bacillus.

A PRACTICAL TREATISE

ON

MEDICAL DIAGNOSIS

FOR STUDENTS AND PHYSICIANS.

BY

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AND TO THE WEST PHILADELPHIA HOSPITAL FOR WOMEN.

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TO THE

MEMORY OF MY FATHER

BENJAMIN MUSSER, M.D.,

AND

MY GRANDFATHER

MARTIN MUSSER, M.D.

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PREFACE.

MODERN methods of medical education demand that the student should be taught the expressions of morbid action, or, in other words, the phenomena of disease. He must be brought into contact with them in the hospital ward and the out-patient room, which are the medical laboratories where all the data are collected, analyzed, and used in discriminating the various disorders.

The object of this volume is to aid the student in the pursuit of such laboratory studies, and at the same time to furnish the practitioner with a reliable practical guide to diagnosis for use in his daily work. It has been thought best to combine in these pages the study of the objective phenomena or *signs of disease*, the subjective phenomena or *symptoms*, and the methods employed for their determination. Special attention has been paid to research for objective phenomena appearing in physical, chemical, and biological changes in the tissues and secretions. The necessity for elaborate descriptions or extended lists of minutiæ as guides to differentiation is being rapidly displaced by the use of instruments of precision. Formerly, for instance, extensive tables were displayed to indicate the differential diagnostic features of anæmia and chlorosis; now a few moments' examination of the blood decides the nature of the affection and whether iron or arsenic is to be given for its cure.

The following pages bear evidence that the author does not undervalue the direct and collateral data obtained by inquiry. Without them an examination carefully conducted according to all other methods may go for naught in the distinction of disease.

The association of morbid processes with their phenomena is a practice of the utmost importance to students, and a chapter has therefore been inserted upon the Symptomatology of Morbid Processes. Bacteriological Diagnosis has become an established method

by which various disorders are recognized, and it is essential that the procedures in this new means of research should be fully outlined. The chapter on this subject is included not merely as a guide and reference for the trained student, but it is hoped that it will also emphasize the possibilities of bacteriological studies and inspire those who are themselves without facilities for prosecuting laboratory work to have examinations made for diagnostic purposes by experts with laboratories at their command.

My best thanks are due to my associate in private and hospital work and teaching, Dr. H. B. Allyn, for assistance without which this book could not have been written; to Dr. H. Toulmin for aid in the collaboration of the sections devoted to the examination of Sputum and Fæces; to Dr. Charles Burr, of the Infirmary for Nervous Diseases, for the articles on Cerebral and Spinal Localization and on Electrical Diagnosis, and to Drs. Joseph Sailer, W. H. Fenn, and J. E. Talley, for valuable assistance.

Fortieth and Locust Streets, Philadelphia,
February, 1894.

CONTENTS.

PART I.

GENERAL DIAGNOSIS.

CHAPTER I.

PAGES

General Observations 17–23

CHAPTER II.

The Data Obtained by Inquiry 24–48

CHAPTER III.

The Data Obtained by Observation 49–144

CHAPTER IV.

Bacteriological Diagnosis 145–158

CHAPTER V.

The Examination of Exudations, Transudations, and Cystic Fluids 159–170

CHAPTER VI.

The Morbid Processes and their Symptomatology . . . 171–186

PART II.

SPECIAL DIAGNOSIS.

CHAPTER I.

Diseases of the Nose and Larynx 187–221

CHAPTER II.

Diseases of the Lungs and Pleuræ 222–337

CHAPTER III.

PAGES

DISEASES OF THE HEART, THE BLOODVESSELS, AND THE MEDIASTINUM 338–429

CHAPTER IV.

DISEASES OF THE MOUTH, FAUCES, PHARYNX, AND ŒSOPHAGUS . 430–466

CHAPTER V.

DISEASES OF THE STOMACH, INTESTINES, AND PERITONEUM . . 467–583

CHAPTER VI.

DISEASES OF THE LIVER, SPLEEN, AND PANCREAS 584–626

CHAPTER VII.

DISEASES OF THE KIDNEYS 627–682

CHAPTER VIII.

DISEASES OF THE BLOOD AND DUCTLESS GLANDS 683–704

CHAPTER IX.

CONSTITUTIONAL DISEASES 705–722

CHAPTER X.

THE INFECTIOUS DISEASES 723–786

CHAPTER XI.

DISEASES OF THE NERVOUS SYSTEM 787–860

MEDICAL DIAGNOSIS.

PART I.

GENERAL DIAGNOSIS.

CHAPTER I.

GENERAL OBSERVATIONS.

The data upon which a diagnosis is based: The data obtained by inquiry. The data obtained by observation.—Object of diagnosis.—Requirements on the part of the student.—Methods of diagnosis: Direct. Indirect (by exclusion). Differential.—Diagnosis sometimes impossible. Avoid haste.—Diagnosis should not be limited.—Modern diagnosis.—Case record.—Scope of the present volume.

THE sufferings of one who comes under the care of a physician are indicated by symptoms of which the patient himself is cognizant, and for which usually he applies for relief; or by alterations of the physical or chemical structure of the whole or a part of the body, or of the functional activity of organs—alterations which, although not apparent to him, are evident to the observer, the physician. The symptoms of which the patient complains, and of which he alone has knowledge, are known as the *subjective symptoms* of disease. The symptoms which the physician observes, some of which, as the changes of the exterior surface, may be apparent to the patient, are known as the *objective symptoms* of disease.

The subjective symptoms of disease, as well as such objective symptoms as the patient is aware of, have a history. It may be the brief one of sudden onset, or a long one of rise and fall, of ebb and flow, of the mingling of complex phenomena from time to time. The story of the evolution of the disease is written as the *history of the present disease.*

The present disease may be due to previous attacks of disease, or be modified by the occurrence of previous disease. We may be consulted for the effects of one link in a chain of morbid disorder which began in early infancy or adult life. We should learn, therefore, of the occurrence of *previous disease.* Certain types of constitution and some few diseases are transmitted by parents to offspring, and, therefore, in a consideration of the patient's suffering we should inquire into the *family history.* A further insight into the nature of the suffering may be

obtained by a knowledge of the age, sex, habits, occupation, environment, etc.—in short, by a knowledge of the *social history*. For, if the cause of the disease under consideration is determined, frequently a distinction from other affections with allied phenomena can be made.

The *subjective symptoms*, the *history* of the *present disease*, the *previous history*, the *family history*, and the *social history* are learned by *inquiry* of the patient or the friends of the patient by means and within limitations hereafter to be described. It is proper that they should be ascertained, if practicable, before the objective symptoms are studied.

After the story of the patient is ascertained in full, the *objective symptoms* are sought for. Examination of the patient by the use of the senses of sight, of touch, of hearing, with the instruments of precision to aid them—the physical examination—and by chemical and bacteriological methods, reveals the presence or absence of the latter class of symptoms.

The phenomena of disease are ascertained, therefore, by *inquiry* and by *observation*. The facts or data thus collected and the discriminate interpretation of them constitute *diagnosis*.

Object of Diagnosis. The object of diagnosis is to determine the condition of the living patient who may be suffering from disease. It implies not only that the phenomena of disease are detected, but also that the effects of the disease on the organism are determined, and that the morbid process which is the cause of the phenomena is ascertained. Even this is too restricted an idea of diagnosis. It should include also the recognition of the cause of the morbid process. The latter is known as the *ætiological diagnosis*.

Diagnosis is not made in order to give a disease a name, but to treat it, and as it is not disease that we treat, but a patient with an ailment, full knowledge of the patient and of his environment, his mode of life, habits, occupation, etc., must be obtained by inquiry.

The practical result of diagnosis is the ability to remove or prevent the occurrence of the morbid processes, or to mitigate their effects by rational therapeutics.

Requisites on the Part of the Student. As data are to be collected by inquiry and by observation, it is obvious that he who would inquire and observe intelligently and successfully must be possessed of knowledge and qualifications of a high order. The phenomena of health must be familiar to him. He must have a full knowledge of physiology to recognize aberrations of function, and of pathology to understand the production of symptoms by disease. He must know the organic results of morbid processes—pathological anatomy. He must have learned by *reading* and *experience* the significance of symptoms or of groups of symptoms and their relation to morbid processes.

He must have a knowledge of the evolution of disease and the phenomena of each period in its development to secure an accurate account of the disease under consideration. He must know the influence of morbid processes on the body and their effect in the production of sub-

sequent disease, in order to ascertain correctly the various diseases of the patient and infer rightly their relation to the phenomena under consideration. The significance of the family history can be appreciated and correctly applied only by a knowledge of the diseases which are inherited or which arise in certain physical types of individuals, which type is inherited. The social history is not worth securing unless the inquirer knows the influence of age and sex, of race, of occupation, of habits, of residence, of degree of labor, in the development of disease, or the influence of the environment on the individual—the action and reaction of external forces on forces within.

To ascertain the *objective* symptoms, he who would observe properly must know anatomy to recognize the seat of disease, and physiology to know the departures from health. He must be trained at the bedside in the use of the senses, and know how to discriminate and interpret phenomena observed by them. He must know how to use instruments of precision, as the microscope, and must learn its revelations; the laws of chemistry and the methods of chemical examination must be familiar to him. Bacteriology and the data derived by its methods must be appreciated fully.

It is thus seen that the inquirer must have knowledge largely gained by reading, by which he acquires the recorded experience of others and learns that certain symptoms under certain circumstances indicate a definite malady, and by observation at the bedside and in the post-mortem room, by which he learns that certain symptoms are associated with definite lesions.

Methods of Diagnosis. But we must not only secure facts, but be able to utilize them for analysis and induction—the result of which is the formation of the diagnosis. The diagnosis is obtained by three methods—the direct, the indirect, and the differential. By the direct method the data collected are sufficient to warrant a positive conclusion. An indirect diagnosis is made by exclusion. A symptom group may represent several diseases. Each affection is passed in review and excluded until one is found to correspond more closely to the data. It is not one, because of the absence of certain symptoms; it is not another, because of the presence of certain essentially different symptoms. A negative is thereby proven. By the differential method the diagnosis of one of a few possible diseases must be made, the data for and against which are passed in review. The direct method is scientific and the most satisfactory.

Diagnosis sometimes Impossible. Notwithstanding our efforts to collect data by inquiry and by observation, we are often unable to make a diagnosis. This arises because premises are wanting in the induction. The subjective symptoms may not tally with the known processes of disease, or the narrator of the history of the present disease may omit important evidence from lack of memory or knowledge, from design, or for other reasons. The objective phenomena may have developed in an ill-defined way, or are obscure, as the state of the abdominal contents in obesity, or they may point to one or more processes the subjective symp-

toms of which are not present. At the time of observation the disease may not have developed fully, may not have "spelled itself out," as in the early stages of the exanthemata. Under these circumstances a provisional diagnosis must be made or conclusions held in abeyance. If we are considering a contagious disease, for sanitary reasons all doubt should be settled in favor of the infectious disease. If, on the other hand, the disease requires prompt remedial action, the symptoms must be taken as the indication for therapy.

Avoid haste. If prompt action is not required, too great haste should be avoided. It is not necessary to make a diagnosis at once, and it is not a confession of ignorance if time is asked before an opinion is given. Repeated observation and reflection should be employed before a conclusion is arrived at. This particularly applies to the class of cases which represent a condition the resultant of an improper environment, for the proper detection of which social data, knowledge of temperament, etc., must be acquired. Then, again, it may be necessary to observe the patient under changed circumstances, or study the effects of diet on renal secretion, or on the function of other organs. Haste leads to faulty diagnosis, and therefore to misdirected therapeusis.

Diagnosis should Not be Limited. It is not sufficient to give a name to a group of symptoms, and be satisfied that the diagnosis is made. Every method must be used to collect data. The exact physical condition of the patient must be ascertained and the functional powers of all the organs correctly determined. We thus learn if the more evident disease is the single expression of a morbid process or if it is the surface storm, the currents of which are underneath. A pleurisy or pneumonia may be the outcome of or complicate a latent nephritis. A peritonitis may be the sequela of an appendicitis or pyosalpinx. Or disease in two or more organs, due to the same process, may exist at the same time, as suppurative pleuritis and pericarditis. It would not be sufficient to recognize the empyema alone.

For purposes of treatment it is not sufficient to recognize a neuralgia or a spasm. The state of the patient on account of which the neuralgia developed must be ascertained. Attention must be called to the importance of not being lulled into a false security by the belief that the diagnosis of the first day is sufficient. Complications may arise or the morbid process invade new territory. Thus, in the course of pneumonia, in a few days a meningitis may arise, or an ulcerative endocarditis ensue.

Modern Diagnosis. Anyone who takes the trouble to recall the methods of diagnosis that were in use twenty years ago will be struck by the wonderful expansion of the means now at hand to unravel the mysteries of disease. Then a few instruments of precision and a few chemical reagents were required. The microscope was employed to examine a few of the excretions and the blood only. Now the instruments of precision are multiplied and the scope of their explorations increased.[1]

[1] As a most simple illustration, witness the knee-jerk and reflexes, learned by an old method, percussion, in extended use.

Chemistry, among other things, helps to fathom the mysteries of gastric disease. The domain of the microscope has increased, and with the new methods of staining fluids and tissues, is the key that unlocks many of Nature's secrets. The new science of bacteriology has come to our aid, and now, before waiting until an epidemic counts its victims by hundreds to establish a diagnosis, it is at once attained.

Certainty in diagnosis, for these reasons, has made a decided advance. The number of diseases which can positively be diagnosticated has increased. Methods of investigation and new instruments of precision are daily on the increase. May we not hope that in the future the horizon of absolute knowledge will be extended far beyond the present? New instruments and new methods will surely avail.

The use of the large number of instruments that are essential, and the chemical and bacteriological examinations that are made, require a great deal of time. Often the diagnosis is a question of hours or even of days. The patient profits thereby. The tax on the physician is far greater than a few years ago. The bedside labor is great, and in addition he must have a laboratory at his command for microscopical, chemical, and bacteriological work. The outcome is that the scientific physician must have a *clientèle* limited in number, or else have one or more assistants to aid in his investigations. Without doubt the latter will soon occur. Not as in days of old will we find in the practitioner's office the apprentice, compounding drugs and rolling bandages, assisting in the operation of bleeding and dressing ulcers, but the highly trained, scientific assistant who by labors in the laboratory and at the bedside is competent to collect data suitable for scientific methods of reasoning.

Case Records. Records of cases should be kept, for many obvious reasons. The habit compels a general survey of the case, and tends to prevent oversight in the examination. It naturally aids in the training of the powers of observation. It teaches precision in the narration of cases. The memory is aided by repetition and by lack of haste in ascertaining phenomena. The data are on record for more mature reflection, and to aid in a study of the literature of similar cases. The record is of value in case the patient returns for advice after a lapse of time. It may be of medico-legal value. The mental effect on the patient is good, for the taking of notes requires time and accurate studied observation. In case it is desired to study a large number of cases, records are scientific data. The records may be kept on loose sheets and filed for future use. When a sufficient number are secured they may be bound in volumes devoted to the respective disease. Or they may be noted in a blank-book selected for the purpose. At the end of the year the book is indexed according to the diseases and the names of the patients. A better method is by a system of cards. The cardboard should be six by eight inches. One card is devoted to each case, although more can be used. They are arranged and catalogued according to the library system of card catalogues.

Method of Record. A systematic plan must be pursued in noting the cases. It need not correspond to the lines of inquiry in the examination of the patient, which are modified by the circumstances of the case.

The social history, the family history, previous diseases, the history of the present disease, including the mode of onset and the duration of the disease, should be recorded in regular sequence. In the history of the present disease the subjective and objective symptoms should be recorded in order. The subjective symptoms that refer to special systems or organs, and the objective symptoms of the same, should be recorded under the special heading.

RECORD OF CASE NO. —

Diagnosis. *Result.*

Name and residence, place of birth, and former residence.

Social history.

Age, sex, race, married or single.

Occupation: Present and previous.

Habits: Tobacco, alcohol, narcotics; sexual habits; regularity of meals, character of food, and method of eating; number of hours of sleep, degree of fatigue; brain use.

Family history: Hereditary tendency; health of parents, brothers, sisters, etc. Cause of death and age at which it occurred.

Personal history: Children, the number and health; miscarriages.

Previous diseases: Character of convalescence; syphilis and gonorrhœa; injuries.

Present disease: Date, mode of onset, and probable exciting cause of present trouble; evolution of the disease to date of examination.

Present condition: Subjective symptoms.

Objective symptoms.

External appearance, development, color, figure, height and weight, attitude, expression of face.

Temperature, perspiration, eruption, swelling. Condition of limbs and joints.

Examination of the *digestive apparatus:* Mouth, tongue, gums, and pharynx; abdominal organs; contents of stomach, fæces.

Examination of *respiratory apparatus:* Nose, mouth, and larynx. The lungs: inspection, palpation, percussion, auscultation, mensuration. Cough and expectoration.

Examination of *circulatory apparatus:* Inspection and palpation of cardiac area; percussion, auscultation of heart; similar examination of arteries and veins, the pulse; examination of the blood.

Examination of the *urinary apparatus:* Kidneys, ureters, and bladder; examination of urine.

Examination of the *nervous system:* Intelligence, subjective nervous phenomena, sleep, gait, station, reflexes, paralysis, tremor, pain, convulsions, headaches, disturbances of sensation, disturbance of speech. The organs of special sense.

Examination of fluids obtained by puncture.

Bacteriological examination of blood, sputum, secretions, exudations, etc.

Diagnosis.

Prognosis.

Treatment.

Scope of the Work. In the following pages the data collected by inquiry and observation will be considered, and the attempt made to show their application in individual disease. Hence, the value in diagnosis of the social history, family history, previous disease, and history of the present disease, will be discussed. The subjective and the objective symptoms of disease and the methods of ascertaining them respectively will then be considered. After the subject-matter above indicated is considered, in a general way the phenomena or symptoms of morbid

processes or of varying causal agencies will be treated of, in order that the student may have a general comprehension of semeiology.

Classification. This is based upon diagnostic convenience. No attempt is made at a scientific pathological classification. Diseases that are not common are described under the objective symptoms of disease in the order of their chief diagnostic condition—as myxœdema or acromegaly under enlargement—or under the structure or organs the subject of objective examination, as myositis under muscles, Raynaud's disease under an account of the extremities.

The student can by ready reference make practical use of the work, as the handbook is used in the laboratory, if he will bear in mind its plan. He first obtains data by inquiry, reference to which can be made under the appropriate section. Subjective phenomena are included in the inquiry. After the subjective symptoms are ascertained, the objective symptoms are looked for. They are arranged in a manner similar to that of the subjective symptoms. Thus, loss of weight will be studied in the consideration of general objective symptoms, contraction of the chest under diseases of the respiratory apparatus. An account of general phenomena, or those which refer to a structure of the surface, as the skin, the eye, or to general structures, as bone, connective tissue, glands, muscle, etc., can be found by reference to the body in general, or to each individual structure, arranged under the objective symptoms. The phenomena which point to an apparatus or system, as pain referred to the chest, for instance, or shortness of breath, will be discussed under the chapters which consider the various systems, as the respiratory or cardiac system on the one hand, or the digestive on the other. It is scarcely necessary to advise the student to consult the index freely.

There is nothing more important to the student than to have a comprehensive view of any subject under consideration. It is recommended that an outline be made of the subject-matter contained in this volume. It can be done in small compass, and if carried in the pocket will be convenient for review at odd times. It is preferable that the student should make the outline himself, hence it is not included in the work. He is recommended to note the subjects as arranged in the index as headings, and underneath them to jot down the divisions of the subject as indicated in the respective portions of the text by the sub-heads, or by antique or *italicized* words.

CHAPTER II.

THE DATA OBTAINED BY INQUIRY.

The Social History: Age, sex, occupation, habits, residence (past and present), family relations, exposure to contagion. *The Family History:* Parents, grandparents, brothers and sisters of each—Brothers and sisters of patient—Wife and children. *Previous Diseases. History of the Present Disease:* Duration and mode of onset—Evolution of the disease. *The Subjective Symptoms:* Their value—Their fallacy—*Feigned disease*—General subjective symptoms—Local—*Pain.*

THE subjective symptoms of the disease are elicited first, so that, by attending to the complaints of the patient measures may be directed promply for his relief; second, that we may have the advantage of observation of the patient's intelligence, expression, etc., and at the same time ascertain the direction further inquiry should take; third, in order that embarrassments may pass off and composure ensue before an objective examination is made. It seems preferable, however, to begin the record with the social history of the case, for a scientific and orderly procession in the data acquired, and then proceed to record the facts of family history, previous history, and history of present disease. Certainly it is immaterial how they are considered in the following discussion, and for convenience, therefore, the above order will be followed. It is to be remembered that the patient's complaints, and the objective phenomena—or if unconscious or otherwise unable to speak intelligently, the latter alone—are the central threads around which the diagnosis is woven.

The Social History.

The aid derived in the diagnosis by the collection of data, by inquiry into the social history, cannot be considered exhaustively. Works on hygiene must be consulted. General ideas will be given; reference to the influence of the various factors will be found under the individual diseases. That such data are of value is seen for instance in distinguishing various forms of colic. Knowledge that the patient labored in lead often will simplify an obscure problem.

The Age is learned, for each period in the evolution and involution of life has its peculiar physiological processes susceptible to variations from external influences. In infancy and childhood the environment is inquired into; at puberty again there is change; in middle life the influences of occupations or habits are felt; at the menopause the blotting out of one function is perturbing; as old age approaches the effects of wear and tear and degeneration and cell-wanderings ensue.

But first, a large group of affections arise in the first period of infancy,

from inheritance or congenital malformations, from accidents incident to childbearing, and from improper management of the cord.

Second, in acquiring adaptability to environment, by the feebly resisting organism, *disturbances of digestion* from poorly prepared or improper food arise; *pulmonary disorders* from improper clothing, ventilation, etc., occur. The developing nervous system has more acute susceptibilities, and hence a long array of reflex symptoms or diseases is observed at this period.

Another group of diseases, the *exanthemata*, and all contagious diseases, are more prevalent in early life, because they arise out of exposure to a specific cause which occurs before the child attains many years. The anatomical arrangement of the larynx, disproportionately small, makes the diseases of it most frequent in childhood, and a serious factor in mortality. At puberty we see the perversions (from earlier years) liable to arise as adolescence advances. Anæmia and chlorosis are very liable to develop at this period. In the middle period, the diseases that arise from occupation, from exposure to external agencies, from habits, are seen. Moreover, processes beginning in adolescence are reaching their acme, and find expression in later life, as the cysts of hydatid disease, or renal calculi, or manifestations of gout. In later life the degeneration of the vascular and cerebro-spinal systems occurs; cancer, affections due to fibrosis, a resultant of wear and tear; calculous disease, and other diseases, prevail.

The Sex. The prevalence of various diseases in the sexes in undue proportion arises because of difference in the anatomical structure and physiological offices of the two, and because of the difference in exposure to varying causal agencies. Diseases of the male sex occur from exposure, by virtue of their occupation, to causes from which the female is exempt, from over-activity of mind and body, and the formation of bad habits. The diseases of the female sex that are more prevalent, apart from their own peculiar affections arising out of menstruation and childbearing, take place because of the more or less sedentary nature of their lives, and hence, among other things, the opportunities for introspection. Hysteria and neurasthenia and nerve disorders abound with them. Males are more subject to epilepsy, gout, diabetes, locomotor ataxy, and vesical disease. Females are more subject to exophthalmic goitre, rheumatoid arthritis, chorea, and the above-mentioned nervous disorders.

Occupation. This must be ascertained in the inquiry, for each occupation demands effort in one particular direction, or compels exposure to deleterious consequences. Writer's cramp, eye-strain, and a series of disorders thus arise. Knowledge of exposure to particular irritants, coal or fine particles of metal or stone, gases, chemicals, effluvia of all kinds, and to diseases contracted from animals, are valuable points in diagnosis.

The manner and degree of employment of the mind must be inquired into.

It is not to be forgotten that the occupation at different periods of life must be found out, the age at which life's battle began, and the circumstances that surrounded the early career. The deleterious influence of

a former occupation may be observed after the patient is in another sphere of labor.

Habits. Habits as to clothing (catarrhal affections and rheumatism), as to hours of rest and sleep (neurasthenia), as to *character* of food, time, regularity, and manner of eating (the indigestions, gout), as to the use of stimulants (cirrhosis of the liver, neuritis, brain affections), of tobacco (amblyopia, cardiac palpitation), of tea or coffee, of narcotics, and as to exercise, must be inquired into. Robbing hours that should be devoted to rest, for labor or dissipation, tells a thrilling story at times; it has an influence on the organism for evil. A knowledge of the habits, of the life—of the inner life, indeed—of the individual, is essential to attain a rational diagnosis, and hence a true therapeusis.

Place of Residence and Dwelling. A knowledge of the place of residence is of service. Town residence or country residence, a residence in a damp locality, by the sea or in the mountains, in particular valleys, in different watersheds, in tropical or frigid clime, makes an impress on the constitution, even if actual disease is not created. Hence malarial regions, goitre districts, localities in which vesical calculi are prevalent, or in which special epidemic diseases abound, as yellow fever, cholera, or dysentery, must be inquired for. Knowledge of the residence at different periods of life and the duration of such, is often important information.

The situation, and degree of comfort for habitation, of the dwelling must be learned. The sanitary arrangements, drainage, ventilation, water-supply, heating, are to be scrutinized.

Family Relations. Marriage, and the number of children, with their degree of health, must be recorded. If a woman, the number of children born, the character of the labor, the number of miscarriages.

Is there trouble in the marital relation? Has there been sorrow or sudden shock, or long nursing, or great care? Are the financial circumstances easy? Has there been recent malfeasance? How many invalid women arise out of such ashes!

Questions so personal can often only be put after long acquaintance, or through judicious inquiry of friends.

More delicate questions must be put frequently, as to masturbation or excessive venery, but with great caution, and only when conditions demand it. In epileptiform convulsions, profound hysteria, neurasthenia, the development of locomotor ataxy, or spinal paralysis, prompt, clear, manly questions as to these habits are to be put, not reference made to them in prudish or mawkish suggestion.

Exposure to Contagion. If the suspected ailment partakes of the nature of a contagious disease, the probability of exposure to the disease must be looked into, and the presence of epidemics ascertained. The period of incubation must be known in such cases. The prodromal symptoms must be ascertained.

The Family History.

The inquiry is instituted in order to determine the affections which may or may not be hereditary. We learn also the average duration of life in the family, and the relation of the mortality to the physiological epochs in life. Data of the latter character is of value in the estimation of the possible duration of life for purposes of life insurance, but also throws light on the recognition of abnormal conditions; thus to learn that most of the members of the family died of apoplexy at a comparative early age, or of aneurism or other arterial degenerations, is to learn that arterial changes developed earlier than usual. To secure accurate data, the age and state of health, if living, of parents, brothers, and sisters are ascertained; or if dead, the cause of death and age at which it took place. Similar questions may be applied to several generations of the family and to collateral branches.

Concerning the question of direct inheritance of disease, but few are strictly so. Of these, nervous diseases are the most common, as progressive muscular atrophy, hereditary chorea, Thomsen's disease, Friedreich's ataxia, migraine, epilepsy, and forms of insanity. The writer has seen chronic Bright's disease, or a state of the constitution that predisposes to it, occur in several generations without the usual exciting causes of that affection. Syphilis is inherited. Hæmophilia is the most striking affection that is transmitted by inheritance. It is not diseases that are hereditary, but types of tissue that predispose to disease, as in tuberculosis, or cancer, or conditions of the organism that favor imperfect metabolism, as is seen in gout or rheumatism.

The family physician, who comes in contact with one or more generations, profits most by the knowledge of the family history. He learns the predisposition to various minor ailments—to headaches and attacks of indigestion, "bilious attacks," for instance; he learns the power of resistance in the family to disease, or its capability to undertake large duties in life; he learns of their susceptibility to drugs, and the tendency in them to take stimulants. Nerve force is the capital with which the battle of life is kept up. If it is at a minimum in groups of families, diseases or conditions of poor health due to its use, a use not excessive in others, arise.

In the inquiry, it may be well to ascertain the probability of disease being transmitted from husband to wife, or the opposite. Syphilis and gonorrhœa, and tuberculosis are examples. Then, too, we must inquire of mothers for the manifestations of syphilis in the children.

Caution must be exercised in the pursuit of knowledge of this kind, as strained, or even ruptured, marital relations may result from injudicious intimations. Not only does it apply to the transmission of disease between husband and wife, but its transmission along lines of families. Caution must be employed in order not to arouse family pride if evidence of "scrofula" is sought for, or provoke undue alarm when inquiry into the family history of cancer is made. Inquire for the symptoms of the disease in various organs in which it may occur, as jaundice, uterine hemorrhage, etc., or ask about growths or tumors. Do not use the specific terms, consumption or cancer.

Moreover, care must be exercised to secure definite data, not to lay stress upon statements of the patient or parent as to the cause of death being "dropsy," or "jaundice," or "cold," or "teething," or "change of life." Control questions must be put by inquiry into the character of the symptoms that attended the fatal illness, and by giving the affections the various popular names that are given them in different countries.

The data of the family history are of no avail unless it is remembered that many fundamental affections have various modes of expression. Various diseases may be allied to the one suspected to exist in the patient, because of this difference of expression. One member of a family may die of heart disease, another of rheumatism, or some have had chorea, or cutaneous affections, or renal calculi; such ailments are expressions of the same morbid process. Finlayson well puts them into groups and fittingly portrays them as follows: "In regard to scrofulous [tuberculous] diseases, we ask for swollen glands or 'waxen kernels,' or runnings in the neck, diseases of the spine and other bones, bad joints, white swellings, or 'incomes' as they are termed in Scotland; disease of the glands, of the bowels, water in the head, consumption of the lungs, or decline, or weakness of the chest with spitting of blood, and so on.

"Heart disease, rheumatism, chorea, psoriasis, and some other cutaneous affections, and perhaps renal concretions and emphysematous bronchitis, appear to replace each other in different members of the same family.

"The neurotic group includes the various forms of neuralgia, epilepsy, hypochondriasis, hysteria, and insanity; apoplexy and hemiplegia may (perhaps doubtfully) be included in this group; their hereditary character seems rather to be associated with vascular disorders. Gout, disease of the liver, contracted kidney, renal calculus and gravel, and angina pectoris form another allied group; and these have also some affinity with the disorders connected with arterial degenerations. Syphilis, which, of course, has marked hereditary characters, assumes such a multitude of forms as to preclude enumeration; but the tendency is for such syphilitic diseases to fail in the course of time from early death or sterility. Abortions, stillbirths, early deaths in infancy associated with cutaneous eruptions on the buttocks, and with snuffles, are important in many family histories; nervous deafness, opacities of the cornea, notched teeth, epilepsy, and imbecility are occasional manifestations of the same disorder in those children who survive."

It is thus seen in securing the family history data are acquired which may be (1) complete and of value in estimating family tendencies; or (2) vague and of doubtful value. The latter occurs because of the want of recollection of matters inquired into, or because of ignorance of the terms employed. The difficulties must be overcome by control questions prompted by our knowledge of the nature of disease and its frequency at different ages, by an inquiry for symptoms, and by investigation into collateral and remote branches of the family.

The fact that diseases skip a generation (atavism) must be remembered. A generation may be small or decimated by accident or acci-

dental disease, and hence the force of the family history be weakened. At times in a family, sufficient time had not elapsed for predisposition to arise, as when the illness of a child is inquired into, the parents of which are in early adult life. Finally, all negative facts must be recorded. Such knowledge must act as a control element in the estimation of the value of the family history.

Previous Disease.

The remote effects of disease, and of its sequelæ, as impressed on the organism, make it essential to inquire into the nature of the previous disease of the patient whom we are studying. The date and character of the disease, the duration, the degree of severity, and the completeness of convalescence must be determined.

Many diseases, as the exanthemata, usually occur but once in the same person, and, therefore, in the diagnosis of obscure cases, if a history of their occurrence has been ascertained, they can be excluded in the count. Others recur from time to time, as croupous pneumonia, chorea, acute rheumatism, and tonsillitis. The history of a previous attack of a certain disease may point to the nature of a second attack which otherwise may be obscure. Some diseases, as rheumatism, syphilis, and gonorrhœa, have pronounced sequelæ. Knowledge of the occurrence of the primary disease may solve doubts as to the nature of the sequelæ.

Infectious diseases lead to forms of neuritis and to brain affections, or to inflammations of organs. The seat of the specific inflammatory process varies in different diseases; after measles we find the mucous membranes impressionable; after scarlet fever, the ears and kidneys liable to inflammation. The history of an attack of hepatic or renal colic may point to the diagnosis of an otherwise obscure process in the respective region.

The history of injury must be sought for in brain and spinal affections. The occurrence of a surgical operation in the past may point to lesions for which it was resorted to, which again may be the source of disease.

The History of the Present Disease.

Scope of the Inquiry. The history of the present disease includes an account of the sufferings of the patient, which I have said are the subjective symptoms of the disease, and of the duration of the disease, of its mode of onset, and the evolution of its symptoms up to the time it was seen by the physician. The patient also gives an account of such objective symptoms as could be noted by him, as swollen legs, the date of their commencement, mode of onset, and progress. In the case record, the history to the date of examination is first recorded, and then the subjective symptoms are noted. The same order will be followed in the text. Practically, it is better to learn the symptoms on account of which the patient applied for treatment, and, with that as a guide, to inquire into the date of origin and mode of development of the disease.

Method of Inquiry. The history and subjective symptoms are best learned in the language of the patient. If the memory fails or the symptoms are not clearly narrated, judicious questions will suffice to complete the story. Leading questions must not be put until the patient's account is fully given.

Often the patient will be too voluble and introduce irrelevant matter, or too taciturn from modesty or a desire to conceal facts, as when illegitimately pregnant. While much time is lost in listening to a prolix or interminable account of sufferings, the student will do well at first to bear with the patient, for it gives him the opportunity to study character, observe the mental and emotional characteristics of the patient and the expression of the countenance. To suppress the loquacious, free the tongue of the silent, gather scintillations of intelligence out of the dense clouds of ignorance, requires knowledge of human nature of a high degree, acquired only by long practice. (Allied difficulties have been discussed in the paragraphs devoted to the family history.) Indeed, the wonderful faculty of seeking information in this manner is the capital of many physicians, past and present, of large practice. It is by this means and by tricks that the charlatan plies his vocation. A favorite method of the quack, after a few words from the patient, is to tell him how he—the patient—feels. They have some knowledge of the march of disease, and portray its full development to the surprised and credulous victim. Elsewhere (see Subjective Symptoms) the reliability of such data is discussed, and the student must not for one moment consider the data obtained by inquiry as of equal value to that derived by observation. The one represents the mere skeleton of the diagnosis.

It is particularly important to secure the chronological order of events of the disease. It is essential and logical, and holds up to clear light the progress of the affection. If such sequence is followed the diagnosis is much easier. Of course, there are circumstances when only the minimum amount, if any at all, of information of this character can be secured. The patient may be unconscious, or in a convulsion, or unable to speak from dyspnœa. It is necessary then to rely on the testimony of friends or to gather it from the circumstances that surround the patient.

Mode of Onset and Duration of the Disease. It is well to learn if the onset of the disease was sudden or gradual. If the former, the most striking phenomena are ascertained—a chill, convulsion, sudden pain, sudden vomiting, a profuse diarrhœa ; each points to lines of further inquiry. If the latter, did it follow upon an acute illness, or did each symptom gradually increase in intensity, and as each week or each month passed by, new phenomena creep into the symptom-complex. We thus learn if the affection under consideration is acute or chronic—its duration. It must not be forgotten that certain affections may be two or three days, or, on the other hand, as many weeks in developing, as typhoid fever, which, nevertheless, is acute. It must be remembered also that diseases may have sudden acute expressions, and that a chronic disease may be in existence a long time without the knowledge of the

patient. An acute colliquative diarrhœa or a convulsion is often the first intimation of a chronic nephritis; an attack of angina pectoris, the first symptom of organic heart disease of long standing. To appreciate the relationship of acute to chronic disease, or of acute phenomena to chronic morbid processes, requires a full knowledge of the processes of disease.

Evolution of the Disease. In making inquiry concerning the evolution of the subjective symptoms complained of, the frequency, duration, character, degree of severity, relation of each symptom to the function of the organ apparently affected, must be inquired into. Thus in the case of pain in the abdomen, we must learn its character, its frequency, its duration, its degree, and its location, and whether associated with functional disturbance of any of the viscera in which the pain presumably has its origin. Or, if there is frequency of micturition, the length of time the symptom was present, the degree of frequency, the time in the twenty-four hours when the micturition is most frequent; its relations to food, exercise, or emotions; the character of the act of micturition, and its association with other evidences of functional disorder in the genito-urinary tract, or of organic changes in the urinary apparatus.

Having ascertained the full story of the patient, including all data obtained by inquiry, special attention must be paid to the sufferings or complaints of the moment. In the manner above indicated they must be further inquired into. It may be they were detailed in the beginning; but information derived by an account of the evolution of the disease or the previous history will require a repetition, with the putting of fresh questions or control questions. Having obtained the chronological account of the factors of life and of disease we are prepared to examine into the significance of subjective symptoms.

The Subjective Symptoms.

The subjective symptoms are expressive of the sensations of the patient, and vary in accordance with the sensibilities of the individual affected. Thus acute pain may apparently represent a severe process in one, while in another the same severity of process may be represented by the minimum amount of pain. It is well known that individuals of one nationality bear pain with greater fortitude than individuals of another.

So, individuals vary not only as to pain sense, but as to other subjective symptoms. The *morale* is shattered in some more readily than in others; thus, for instance, oppression of the præcordia may strike terror and be an alarming sign to some, while to others it would be simply a sense of discomfort. Moreover, subjective symptoms are constantly before the patient, if only in the mind's eye, while in distress, and, because of his perturbed state, grow in magnitude rather than lessen. We must study them from many points of view. The mode of onset, frequency, degree, and character of the symptoms must be inquired into. The competency of the witness under the circumstances, from

lack of accurate noting of symptoms, failure of memory, varying degree of susceptibility to impressions, etc., may well be doubted. But not only does the varying "personal equation" of the patient render subjective symptoms fallacious; the same factor in the physician contributes to the fallacy. The latter may have unfortunately formed by previous hearsay regarding the patient a preconceived notion of the nature of the disease; or from personal bias in favor of particular diseases, on account of narrow lines of study or lack of breadth of view of pathological processes, he sets out to prove a theory rather than establish a fact. In either case, by leading questions, by placing emphasis on certain parts of the testimony, the subjective symptoms can be juggled with and made to tell any but the truthful story.

It is to be remembered that it is not only our province to ascertain the cause of suffering in the sick, but also to detect the flaws in the testimony of him who would feign sickness. The malingerer utilizes subjective symptoms, because they cannot be seen, felt, weighed, measured, or ascertained by hearing, to hide his deception.

Feigned Disease. To detect feigned sickness implies much acumen on the part of the physician. He must not only be able to make an accurate and exhaustive objective examination of the patient, but be alert to appreciate surroundings and conditions. Feigning may be suspected if there is a motive, as in the case of prisoners, pension applicants, students at school or college, persons who hold policies of insurance indemnifying in case of sickness. If sickness recurs frequently without definite cause, the subjective symptoms of which are mild and quickly recovered from, and in which the objective symptoms are negative, it should be looked upon with suspicion. The hospital "beat" thus plays upon charity. The use of instruments of precision will detect the malingerer. By them it can be found out generally if the subjective and objective phenomena tally. The absence of such tally proves the deception. The thermometer frequently exposes the deception, as fever can rarely be simulated, although tricks with the thermometer may be carried on. A favorite method is to rub it, and thus cause the mercury to rise. Frequently the suspected person must be placed under close surveillance, unknown to him, and tricks of all sorts, suggested by the surroundings and circumstances, played upon him to make him unwittingly testify to his deception.

The student will learn later that there is a mimicry of disease, and that in certain nervous affections the simulation of subjective symptoms is its chief rôle. In hysteria, subjective and objective symptoms are marked. Long experience and acumen are acquired by the physician to unmask the deceptions. The age of the patient, the sex, the state of the emotions, the varying expressions of the symptoms (under varying circumstances) with attention fixed or removed, the mobility of the symptoms under excitement or emotional disturbance, the lack of harmony of functional disorder and organic change, are elements to be considered in order to fathom the mysteries. Often anæsthesia must be induced in order to dissipate simulated tumors, relax rigid joints or contracted limbs. Magnetism, electricity, and other tests are likewise

employed. In the chapter on Hysteria its manifold expressions will be adverted to, and it will be seen that functional disorder of almost every organ or special sense is simulated in this affection. Organic processes even are imitated, as joint inflammations, peritonitis, etc.

Notwithstanding the fallacy of subjective symptoms in that they may be feigned or mimicked, they are valuable evidence at the hands of the scientific inquirer. If the patient is a good witness their value is much enhanced. He must be intelligent and truthful. His testimony is of value if he can array in logical order the sequence of symptomatic events which culminated in the condition for which he seeks relief. If he can clearly narrate the events in his past life, or in the lives of his ancestors, which appertain to physiological aberrations, his story is an aid to the searcher for truth.

If, with this, the doctor is possessed of a scientific turn of mind, considering evidence without allowing previous conceptions to influence him, capable of discerning the truth and discarding the false, of analyzing and weighing statements, and of appreciating their relationship to that which is known of morbid processes, the patient's statements of subjective symptoms are of value in the discernment of disease.

The Nature of the Subjective Symptoms. The symptoms of which the patient complains may be general or local. The former will be briefly considered in this section; the latter will be discussed in the respective sections devoted to disease of the various organs to which the subjective symptoms refer. They are symptoms due to functional disturbances of the respective system that is the seat of disease, as dyspnœa or cough in diseases of the respiratory system, anorexia or nausea in diseases of the digestive system. An exception will be made in the case of *pain*. While there may be such general suffering as to constitute pain (general soreness, aching, rhachialgia), yet the symptom has its point of origin most frequently in some local disorder. Notwithstanding this fact, however, as it is a symptom common to so many affections, and as general rules apply to the recognition of its multitudinous forms, a brief section will be devoted to its study.

General Subjective Symptoms. The general subjective symptoms, that is, the normal and disagreeable sensations which extend more or less over the whole body, or are referable to more than one organ or apparatus, are few in number and are not diagnostic of any particular affection. They are at times the only symptoms complained of by the patient, and require investigation, in order to give relief. They include abnormal sensations of strength or weakness, general numbness or tingling, and general paræsthesia of all kinds; general vasomotor disturbance, causing sensations of heat, as occur in flashes, or sensations of cold, from mild chilliness or "creeps" to the pronounced chill or rigor, sudden perspirations, general throbbings or pulsations, and general discomfort, to which the term *nervousness* is applied. Irritability, disorders of sleep, and the more distinct nervous manifestations above mentioned will be referred to in the sections on nervous disease, and particularly discussed under Hysteria and Neurasthenia.

A feeling of *strength*, or the idea of an ability to perform great feats of strength or endurance, or a great mental feat, is a subjective symptom that is dwelt upon by the patient who is about developing or passing through certain stages of paretic dementia. It is accompanied by other evidences of exhilaration. *Exhilaration* attends chlorosis and forms of hysteria and neurasthenia, the physical or mental exhibition of strength taking place in the after part of the day and evening, or upon undue excitement. Corresponding depression usually follows.

A sense of *weakness*, or *exhaustion*, or of *fatigue* is often complained of. If an absolute demand is made upon the body strength it can respond, but otherwise it is not exerted. The patient complains of being more tired in the morning than upon retiring, or of a sense of inability to perform accustomed or special duties. Mental depression usually attends the phenomenon. It is due to neurasthenia generally, but is a frequent accompaniment of and dependent upon the forms of toxæmia to which malaria, gout and rheumatism belong; of the toxæmia of certain varieties of indigestion, of tobacco, alcohol, and other narcotic poisons (tea or coffee) and of mineral poisons. The same sense of fatigue attends the prodromal stage of the specific fevers. It has been a symptom observed frequently of late in the sequential period of influenza.

The sensation of weakness must not be confounded with true weakness or *muscular prostration*. While the patient is aware of its presence, it is well to consider it under the objective phenomena of disease, for it is a readily recognized sign of disease.

Numbness, or *tingling*, or *burnings* may be general or local. It is a common form of *paræsthesia*, to be discussed in the section on nervous diseases. It must be remembered that, while a disorder of sensation, it is due to morbid conditions outside of the pale of the nervous system. It may be of reflex origin, from irritation at a distant point, or it may be and usually is due to a toxæmia, as lithæmia. Other subjective vasomotor disturbances that are of frequent occurrence are likewise manifestations of nerve disorder from reflex or toxic causes. Flushings, or a constant sensation of *heat* with or without *perspiration*, which attend the perturbation of the menopause, are common in uterine disorders and in chronic gastritis.

The student will learn that the curious manifestations to which reference has been made are generally all evidences of ill health, of a depressed vitality, of a condition in which there is malnutrition, poverty of nerve force, and lack of blood richness (anæmia). There may be peripheral irritation or a toxæmia, but the under-current of ill health is the fundamental derangement.

Chill and *fever*. Both are subjective as well as objective phenomena, but as one can be accurately estimated by an instrument of precision (thermometer), and as both are generally associated, the discussion of them will be postponed. (See Objective Signs.)

The abnormal sensation of cold or of heat will be discussed in the chapter on Nervous Diseases.

Pain.[1]

Definition. Pain is a general term used in medicine to describe a number of subjective symptoms connected with morbid processes. It may be defined as the expression in consciousness of injury to the peripheral nervous system, provided the injured part is in connection with the seat of consciousness, the brain (Payne), or it may, in general, be defined as sensation received by the perceptive centres from the afferent conducting paths, which sensation produces on the part of the organism, as a whole, the desire to abolish or escape from it. This definition, however, fails to include the hyperæsthesias, the hyperalgesias and all simulated pains. But the latter are to be included in this section, on the ground of clinical convenience; whilst the two former are only of significance as conducing to the causation of pain.

Pathology. The pathology of pain is generally believed to be a state of impaired nutrition, and hence of injury, gross or microscopic, of some portion of the afferent nerve tract. The cause may be purely functional, as, for example, when pain is due to the over-stimulation of the tract by its normal stimulus, and its consequent exhaustion; or to strictly local conditions, as pressure, injury, or inflammation; or to systemic conditions acting locally, as the neuralgias of anæmia. There is also the so-called sympathetic or reflex pain, due to irritation in a part removed from the locality to which the sensation is referred. In certain cases of neuralgia the nature of the disturbance has not been ascertained.

Variations in Disease. Pain is, perhaps, the most variable symptom in disease. It ranges from a sensation of mere discomfort, as the dull ache of chronic lumbago, to the stabbing pain of pleurisy or the intolerable anguish of heart-pang. It is at times compatible with the highest mental endeavor or the severest physical exertion, or it absorbs the whole energy of the organism in resisting it. It may be definitely localized in any part of the body, in any of the tissues, or distributed over an ill-defined area.

The Recognition of Pain. The Mode of Expression. As a rule, the physician learns of its existence by communication from the patient. Thus he learns more or less accurately its location, character, degree, and duration; and usually something concerning its causation. But the value of this source of information is variable. The patient may be voluble and describe too much; or taciturn and admit too little; or ignorant and unable to give a clear account. Fortunately, there are other ways by which suffering is expressed. (*a*) *Facial expression*, the most common interpreter of the emotion, is far more reliable. The tense and drawn lineaments, the clinched jaws, the dilated pupils, the livid countenance, the labored respiration, the general shrinkage of the body, make an unmistakable picture of agony, Or, in a less intense form, the shrieks and struggles or the groaning of more prolonged suffering are no less impressive in their suggestiveness. (*b*) Not less

[1] Pain is treated of in a suggestive manner and so much space given to it because it is too frequently improperly managed. Its cause is never thoroughly investigated. Anodynes are given for its relief, thus too frequently creating victims of the morphine, chloral or other vicious habits.

characteristic are the various *postures* assumed; the sudden fixity of heart-pang; the retracted head of meningitis; the immobile side of pleurisy; the crouching attitude of cramp; the flexed thighs of peritonitis; or the bent knee of arthritis. (*c*) Further, there are certain *reflex actions* that are associated with local irritations; thus the closure of the eyelid on irritation of the conjunctiva, the sneeze or cough on irritation of the nasal or laryngeal mucous membrane, the erection following irritation of the urethra, or even the limp characteristic of pain on moving or resting the weight of the body on an affected limb. Then there is the sudden shrinking of the whole body, the attempt to defend, or the sudden movement of the hand to, the affected part, or the sudden jerking away of the part itself if the act be possible; these are true reflexes and sufficiently diagnostic of local suffering. It scarcely need be mentioned that in children, in the insane, in persons unable for many reasons to communicate their thoughts, the expression of pain is of the greatest diagnostic value, as to the determination of the seat of pain. (*d*) The associate phenomena of morbid processes may serve to indicate the occurrence of pain and its seat. Thus pain is one of the cardinal symptoms of inflammation; it is commonly associated with nerve injury; it is frequently accompanied by local flushing in neuralgia.

Sources of Error. In estimating the presence or absence of pain, or its degree, certain control conditions must be borne in mind. Unfortunately *pain* is one of the most *unreliable* of symptoms. It is necessarily a subjective symptom, with, in all probability, qualitative as well as quantitative variations. The particular degree in either respect is of importance in diagnosis, and as only the roughest means, if any, are available to estimate it objectively, the physician is compelled to rely almost wholly upon the statements and appearance of the patient. His statement can err in two directions; the patient can *exaggerate* his sufferings or *depreciate* them. The tendency to exaggeration is most marked in the nervous temperament, in those suffering from chronic disease of long standing; in those accustomed to indoor and mental labor; in women, and in the young. The tendency to depreciation is most marked in the phlegmatic temperament; in those accustomed to hardship, especially if of small intellectual development; in men; and in the aged. Both tendencies are to be corrected as nearly as possible by observance of the associated symptoms, and the character of the patient, and by skilful questioning. The appearance can deceive because of undue susceptibility to suffering on the part of the patient, or unusual inhibitory power. There can be no question that painful stimuli, normally easily borne, in some produce almost unbearable misery. Such exaggerated sensibility occurs in the emotional, in the weak and debilitated, and in the delicately nurtured. *Mental association* is a powerful factor; it is well known that soldiers, who in the heat of battle disregard serious and necessarily painful wounds, will suffer intensely under the probably less painful offices of the surgeon; and unfortunately it is a common experience that the surroundings of the operating-room make the most trifling and briefest operations full of serious suffering. *Habitual* use of *opium* seems in a remarkable manner to increase this susceptibility. Patients will even submit to operations for the relief of a supposed ail-

ment that is found to have no physical basis; and this occurs in cases where there is no reason to believe that the pain is simulated as an excuse for the indulgence. *Inhibition* is a much more serious source of error, for while undue attention to one part is only reprehensible when practised to the neglect of others, a patient who disregards pain may fail to direct attention to the real seat of disease. It is sometimes exercised to a most remarkable degree. The stoicism of the American Indian under torture is attested by many observers; certain religious sects among the Hindus habitually afflict themselves in the most ingenious ways; the early Christian martyrs rejoiced in misery. It is common to note this disregard among those exposed by occupation to discomfort and injuries, and the Teutonic and Slavic races appear to possess it in a higher degree than the Celtic or Semitic. *Shock* either inhibits pain or diminishes the normal response to it. Lastly, and by no means to be neglected, a most common source of error is due to undue credulity or skepticism on the part of the physician; for he may be deceived by an eloquent and persuasive complaint or discredit true suffering.

Simulated pain (see Feigned Disease) is to be recognized by the presence of an object. The simulation is common enough nowadays, in those who seek damages for injuries, or in those who have a morbid craving for sympathy and attention. The detection of this depends upon the skill of the physician, for, with ingenuity, by distracting the attention from the part complained of, he observes that the pain disappears, or pain is admitted in a part to which attention is directed; moreover, the physician observes an absence of adequate physical alteration, and usually inconsistency in the symptoms, for rarely is the malingerer able for any considerable time to act a correct clinical picture. Especially in the latter case is the observation of the surroundings of the invalid of considerable importance. The so-called hysterical mask is of much value; the bitter complaints and the placid or even smiling features cannot fail to strike the observer by its incongruity. True hysteria is apt to be deceptive, and more than one humiliating failure is recorded, of even the most skilful of our craft. The difficulty is increased because true physical changes occur, as amaurosis with dilatation of the pupil, contracture and induration about the joints, unquestionable anæsthesias and palsies. It is often to be detected only after prolonged and painstaking study of the case, the careful exclusion of organic visceral disease, the absence of the characteristic symptoms of the nervous degenerations, such as ankle-clonus, or altered electrical reactions, or changes of the fundus oculi, and often by the impossibility of associating the sensory lesions with the known anatomical distribution of the nerves.

Objective Investigation of Pain. In order to estimate accurately the diagnostic value of pain, the statement of the patient must be corrected by his expression, posture, and manner, and the apparent nature of the disease. Pain is one of the cardinal symptoms of inflammation; vasomotor and muscular disturbances are often associated with neuralgia; any morbid condition exerting pressure on a nerve trunk, as a neoplasm, callus, etc., commonly causes pain. Hence, if the objective phenomena of these dis-

orders are present they lend color to the complaint of pain, and, if not complained of, they should be inquired for. Attempts have been made to introduce scientific accuracy into the estimation of the degree of the sensation of pain, or at least to secure a practical method for measuring its varying intensity in different localities in the same case. Björnström, of Upsala, has contrived a pair of forceps that compress a fold of skin; the amount of pressure required to produce pain, which can be read from a scale, indicates the degree of sensibility or rather resistance to painful impression. Another instrument, Buch's, accomplishes the same thing by direct pressure, and hence can be used over the superficial nerve trunks. Another method more generally available is the application of an induced current of variable strength; single, naked-wire electrodes being best for this purpose. And the common clinical method, by far the most inaccurate and only applicable in considerable degrees of analgesia, is a pin or needle forced through a fold of skin. No method has yet been suggested for even the approximate estimation of the degree of internal pain, and it must still be left to the judgment of the patient.

The Clinical Value of Pain. The presence of pain is recognized by the above-mentioned circumstances. Its degree, with the limitations indicated, has been estimated. Its clinical value is then to be considered. From what has been said above, the converse of many of the propositions is true. By pain and the mode of its expression we can judge of the character, temperament, and nervous susceptibility and perturbability of the patient. It aids us in the recognition of hysteria and helps to detect the malingerer. We learn the capability of resistance of the patient, and hence in a measure of his strength. We learn of the receptivity in consciousness of the peripheral irritation. The degree of intelligence, or the amount of stupor, is thus recognized. Or, if conditions are present which usually cause pain, its absence may show disease in the conducting paths to the brain. Further, the absence of pain under the above circumstances points to the occurrence in the local process of such change as has destroyed peripheral nerve-endings. Thus, when pain ceases in dysentery, gangrene has ensued. In intestinal obstruction, its cessation indicates the same process. In profound shock, pain is not complained of; the amount of pain, therefore, tells of the degree of shock. Hence, in peritonitis, in which shock frequently occurs, pain may be wanting entirely. The abdominal surgeons welcome its occurrence after an operation.

While the above lessons, from the presence or absence of pain, are not to be underestimated, the value of pain to the physician is from the standpoint of diagnosis.

By this symptom we may be enabled to determine (1) the location of disease, and judge (2) of the nature of the morbid process on account of which it is excited. The location of the disease is judged (*a*) by the seat of the pain and (*b*) in part by its character. The characteristics by which pain is recognized (see p. 35) also indicate to us its point of origin in a general way, and its probable cause. They are (1) the facial expression, (2) the position, (3) the reflex actions, (4) the associate phe-

nomena. They need not be referred to again. The nature of the morbid process is judged by the study of pain from various standpoints. Thus, in the case in which pain is complained of we must learn, first, the mode of onset; second, the duration; third, the time of occurrence; fourth, the character or variety of the pain; fifth, its seat; sixth, its variability as affected by pressure, temperature, rest, motion, posture, electricity, drugs, and climate.

1. Mode of Onset. The *mode of onset* of the pain is in the majority of cases an indication of the acuteness of the morbid process. *a.* The onset may be sudden. 1. In gout or acute inflammations of serous membranes, as pleurisy or peritonitis, pain may occur suddenly. 2. It is sometimes of sudden occurrence in certain headaches, particularly of congestive or emotional origin. 3. When pain occurs suddenly it is due, further, either to sudden obstruction of parts that are sensitive, or to effort on the part of the structure to remove a foreign body, as in the intestines, the gall-ducts, the vermiform appendix; or in the respiratory tract, the nares, or the bronchi; in the genito-urinary tract, the ureters, bladder, or uterus. 4. Moreover, sudden pain may indicate rupture of the structure in which it is developed. Here we have the most typical sudden pain. Thus, in rupture of an aneurism or of the heart, there is sudden sharp pain. In rupture or perforation of the stomach, of the intestines, or any of the hollow viscera, this character of pain arises. 5. Sudden pain also occurs in certain neuralgias or neurosal affections. It is seen in its most striking form in angina pectoris, and in sudden brow-ache, or trigeminal neuralgia. *b.* Pain that develops gradually indicates that the process is one of gradual development and not attended by a "solution of continuity," as from rupture or tear. It is the pain that usually occurs in various forms of rheumatism, in inflammations of muscles, and of mucous membranes, in slow inflammations of serous structures, and in chronic bone disease.

2. Duration. From the *duration* of the pain we learn of the acuteness or chronicity of the morbid process on account of which it is generated. *a.* Pain of short duration is seen in the affections in which it develops suddenly (see Mode of Onset), in acute serous inflammations, and in neuralgias. *b.* Pain of long duration, if constant, is usually due to organic lesions; if intermittent it may be due to neuralgia. Pain that is continued over a long period of time excludes the sudden accidents that were previously mentioned, unless change in the character of the pain takes place. Pain is also divided, as to duration, into temporary and constant pain. *a.* Temporary pain indicates an abeyance or relief of the morbid process, while the constant pain points to its continuance. *b.* Constant pains are seen in bone affections, in inflammation of muscles, in reflex pains due to chronic disease elsewhere, as the backache of uterine disease, or the inframammary neuralgia from the same cause. Pain may also be intermittent, remittent, and paroxysmal, or periodic. *a.* Intermittent and remittent pains are characteristic of neuralgias, or point to a functional origin, recurring because the cause which superinduces them is again operative. Thus headaches due to eye-strain may be intermittent

or remittent in the sense that they only occur when the eye is used. Such pain continues over a long period. *b.* Paroxysmal pain is the form which occurs when there is obstruction of channels, as the gall-ducts in biliary colic; the intestines, the uterus, and the ureters in the various forms of colic to which they are liable. The paroxysms of pain recur in the course of the attacks. *c.* Periodic is applied to pains that occur at distinct intervals. Pain that is periodic has frequently for its cause malaria in some form. The toxic headaches and nerve headaches, as migraine, are often periodic. (Consult Headaches.) Pain that attends definite states of exhaustion which occur periodically, as at the menstrual period, is of this type.

3. The Time of Occurrence. On inquiry as to pain and its characteristics some evidence of diagnostic value is derived by knowledge as to the time of occurrence of the pain. Pains may occur only in the daytime, or only during the night. Nocturnal pains are common in syphilis. They are usually due to periosteal inflammation, and occur after the patient is in bed. Pains that are limited to the day are usually reflex pains from functional disorder. Some pains, as headache due to cardiac weakness, and forms of anæmia, are present during the day, because the patient is in the upright position. They disappear in the recumbent position, and hence are not present at night.

The time relation of pain to functional acts is of importance. Thus in gastric pain, its relation to the taking of food is ascertained. Pain coming on before meals is gastralgic; occurring after meals, it is due to ulcer or cancer, sometimes to indigestion. So we inquire of chest pain, is it increased by exertion?

4. Character. Pain may be sharp, lancinating, stabbing. Pain of this character is usually due to inflammation of serous membranes, to colic in various forms, and to forms of neuralgia; cutting pain is a sharp form that occurs in flatulent colic. Throbbing pain is usually associated with acute inflammation, whether superficial or deep. It may be rhythmical with the pulsations of the heart. Dull pain is due to slow chronic inflammation in the bones and in the viscera; it is the pain of myalgia and of fatigue in the muscles. It may be of an aching character. But aching pains may also be general; they are found among the prodromata of the acute diseases, attend and follow a chill, and occur in most characteristic form in influenza and dengue. *Pressing* pain is complained of when pain attends an attempt to remove material from the viscera, as the passage of water when the bladder is inflamed; passage of fæces in dysentery; the passage of clots or other material from the uterus is attended by pain with pressure or bearing-down sensations. The term tenesmus is applied to it, so that we have vesical tenesmus and rectal tenesmus.

Finally, the character of pain is often an indication of the nature of the disease as well as of the tissue affected: 1. Thus, the bone and periosteal pains are boring and constant. 2. In muscular affections there is soreness or aching. 3. In the serous membranes the pain is sharp and stabbing. 4. In the mucous membranes, dull and burning. 5. In the skin, burning or itching. 6. In the viscera, dull and usually steady, although in malignant disease of the various organs it may be

sharp and paroxysmal. 7. Aching, burning, and throbbing in the nerve trunk and its distribution, with tenderness, commonly indicate neuritis. (See "pain crises," page 43.)

5. LOCATION. This is, in general, an indication of the location of the disease. It may be accepted as an almost universal rule that pain due to a local process is limited to the immediate or associated nerve supply of the diseased region. This holds true even when the referred pains, that is, those felt in the associated nerve supply, are as far distant from the site of the morbid process as the knee pain of coxitis, the shoulder pain of hepatic disease, or the ear and temporal pain of lingual carcinoma.

It may be of questionable advantage in some cases that the localization of pain generally indicates the situation of the morbid process. Too often an explanation of the symptoms, apparently adequate, may thus be found, whilst other pathological changes may be overlooked. But on the contrary, the condition to which attention has been called by the pain might, on account of its obscurity or unusual location, altogether escape observation.

In the first place we determine whether pain is *general* or *local*. 1. *General pains* are due either to central or peripheral disturbance of the nervous system by a poison circulating in the blood. This may be the poison of fevers, or may be a rheumatic or gouty poison. It is seen in the common affection known as "cold," when the pains are probably myalgic. In syphilis, malaria, lead-poisoning, and toxæmias generally, there is general pain, soreness, and fatigue. General pains are not confined to the muscles, but are also seated in the fibrous structures and bones. In their more severe forms such pains occur in dengue, and are known as "break-bone."

2. *Local pains* may be (*a*) superficial or deep-seated; (*b*) they may be limited to a *small* area or *radiate* in various directions. *a. Superficial* pains are due to involvement of the superficial nerves distributed to the skin or to the muscles directly underneath or to the structures in close relation to the skin, as the peritoneum, the pleura, or pericardium. *Deep-seated* pains, when in the extremities, are due to bone disease; when in the abdomen, to disease of the viscera, particularly inflammatory affections; when in the chest, to disease of the aorta and mediastinum. The diagnostic value of these forms of pain can readily be appreciated. Thus, when pain is complained of in the abdomen, if superficial, it is due to the nerves and the muscle or to the peritoneum. If deep-seated, it may be due to inflammation along the vertebral column, to cancer or ulcer of the stomach, to aneurism, to disease of the pancreas, or of the liver. In the lower portion of the abdomen it is due to pelvic or renal disease. (See Abdomen.) In the same manner, in the chest the superficial pains are due to affections of the walls of the thorax, or of the serous coverings of the lung or heart. The causes of deep pain have been mentioned.

b. The area. In studying the localization of pain we inquire whether it is circumscribed, diffused, or radiating. *Circumscribed* pain is always due to a small area of disease, or is reflex. Thus, in ulcer of the stomach the pain is usually circumscribed to a small area in the epigastrium; in

inflammation of the appendix, to the region of that structure. *Diffused* pain indicates involvement of a large area with less intensity of process than when circumscribed. Pains that are *radiating* are usually distributed in the area of the nerve distribution related to the point of origin of the pain. We learn much from the study of this distribution: the pain of cancer of the anterior portion of the tongue may be chiefly complained of in the ear; the pain of disease of the hip, at the knee-joint; of the liver, at the shoulder. The pain of angina radiates down the arms; of renal disease, to the head of the penis or to the testicles. In diaphragmatic pleurisy the pain is referred to the front of the abdomen above the umbilicus. *Peripheral pains.* Radiating pains, however, are chiefly due to disease in the course of the nerve, the pain being referred to the trunk and terminal distribution of it. In disease of or pressure upon the nerves as they go out from the spinal canal, pain may not be complained of in these situations, but at the periphery of the nerves at the centre of the abdomen. Pain over the abdomen is frequently an indication of disease of the vertebræ, propagated by the sixth or seventh dorsal nerve. Pain between the shoulders is often due to aneurism with pressure upon the vertebræ. (See Pain in the Heart.)

Hilton lays down the rule that pain in any part, in the absence of local inflammation, is due to exalted sensitiveness of the nerves of the part, and depends upon a cause remote from the painful area. The term *sympathetic* is applied to this group of pains. Further, Hilton remarks that pain on the surface of the body must be expressed by the nerve which resides there, and somewhere in the course of its distribution between the peripheral termination and its central origin the cause of the pain must be situated. This applies particularly to the pains which arise from disease of the vertebræ. To the same class belongs the pain on the inner side of the knee in hip disease; at the extremity of the urethra in disease of the bladder; in the testes and thigh in renal calculus, and at the tip of the shoulder in affections of the liver. Pain in the phrenic nerve, in the neck, may be due to pericarditis or diaphragmatic pleurisy. For the same reason pain over different areas of the scalp should be investigated, for often a localized pain is due to disease of the fifth nerve somewhere in its course. In a similar manner pain in the legs is frequently due to cancer of the rectum or bladder. In ulcer of the rectum, pain, cramps in the legs, numbness, and even loss of muscular power, are sometimes confined to the left side only. The same lesion causes pain in the lumbar region, as well as in the limbs. Hilton describes a case in which pain over the sciatic nerve, over the left hip and loin, and over the right leg, was due to a small ulcer in the anus, the curing of which caused relief from the pain. As a corollary to this, in the investigation of the cause of pain, the nerve, its connections, and the organs supplied by it, should be investigated. *Bilateral, symmetrical,* and *superficial* pains indicate a central or bilateral cause; while, on the other hand, *unilateral* pain implies a seat of origin which is one-sided.

Peripheral Pain of Central Origin. In addition to the class of cases, which are of peripheral origin, we must refer to the pains in the extremities or in the trunk that are due to central disease. In

meningitis and other general organic affections of the brain and cord, peripheral pains are most common, and they may be the earliest and most striking symptoms. Indeed, it is most common to find patients with spinal disease to have been treated for a long time for what was supposed to be rheumatism. The pains of central origin in the joints may be constant, or may arise in paroxysms and be of a lancinating character when the disease is chronic. (See Character, page 40.) Severe paroxysms of pain may occur under these circumstances and be most excruciating, sometimes causing collapse. They are known as painful *crises*. In addition to the joints, pain may be complained of in various viscera. Sudden, intense pain, with functional disturbances of the affected viscera, occurs independently of any lesion of the part or of any apparent exciting cause. One class of the attacks is known as *gastric crises*. The pain is in the epigastrium, and is associated with vomiting. In another class *laryngeal crises* occur with pain in the larynx and violent spasmodic cough, with dyspnœa. The pain extends over the shoulders. Or we may have *rectal crises*, with sensation of burning in that situation ; *urinary crises*, simulating renal colic, and *genital crises*. Pain in the muscles, like crises, also occur. Crises occur chiefly, if not entirely, in locomotor ataxia. They are distinguished from the pain of other causes by their sudden onset, their extreme severity, the absence of organic disease or local cause in the affected viscera, the sudden termination, the normal condition between the attacks.

Pain in the joints and in the periphery of the extremities is of frequent occurrence in neuritis ; inflammation of the sciatic nerve may cause pain in the extremity of the foot. Pressure inflammation on that nerve may also give rise to distal referred pain.

6. Pain Modified by Pressure, Movement, Rest. We also study pain under the influence of pressure, movement, temperature, rest, etc. Pain that is modified by *pressure* is generally superficial. It is usually of an inflammatory origin. The variety of the pressure gives some clue to the nature of the pain. If pain is increased by pressure of the finger tips it is due to ulcer or inflammation, when internal, and to inflammation if external. Gastralgia and colicky pains in the intestine, which may be neurotic, are relieved by pressure, particularly if the whole hand is applied. Pain that is due to dislocation of some organ, as movable kidney or displaced uterus, or to dependent viscera, may be relieved by judicious pressure in the proper direction so as to relieve the displacement. The pain that arises from affections of the nerve trunks can be distinctly localized by pressure in the course of the nerve trunk, and particularly at the points where the cutaneous filaments of the nerves come through the fascia. These points are along the vertebral column, in the axillary region, and anteriorly about the parasternal line, in general. By determining the presence of these tender points we distinguish neuralgias from myalgias. Pain due to bone disease can frequently be distinguished in this wise. By pressure or weight upon the head or shoulders we may ascertain whether pain is due to vertebral disease.

Pains increased by *movement* point to an affection of the bone,

muscle, joints, or nerve in the part moved, as a limb; groups of muscles may be isolated for the tests. Some few pains are relieved by movement of the body, only because the mind is diverted in this act. Pain increased by movement is due to myalgia or rheumatism, when superficial.

Almost all pains are modified by *rest*. Its influence has but little diagnostic significance. In some cases of doubt as to the nature of a visceral pain, functional rest of the organ, by which relief is obtained, may aid in determining its locality. Thus, rest to the eye may relieve a headache, the nature of which was obscure until this respite was secured. Pain modified by *temperature* (heat or cold applied to the spine, hot water in a sponge) and by electricity, usually gives information as to the seat of disease in the spinal column, of which the pain is the external expression. Pain modified by climate is rheumatic or neuralgic; modified by weather or season, is due to neuralgia or neuritis, whether of gouty or traumatic origin. (See papers by Weir Mitchell.)

Résumé. Notwithstanding clinical investigation we may not be able from the characters and locality to determine the real cause of the pain. In general it may be borne in mind that pains are due to (1) disease of the central nervous system or the nerve trunks; (2) to inflammations; (3) to intoxications, as from malaria, lead and other forms of toxæmia; (4) to pressure on the nerve trunks; (5) to reflex influences. If in doubt, therefore, the general symptoms and condition of the patient must be ascertained in order to determine the causal origin and hence the true nature of the pain. In all cases of pain the controlling motive in diagnosis should be to determine the *general condition* of the patient and find the *cause* of the pain.

Reference must be made to the curious change that takes place in the chronic intoxication of morphine. Persons with the morphine habit are very liable to have functional pain. This form of pain usually is paroxysmal and severe, and may simulate organic pains. The most common clinical form seen is gastralgia. The subjects of locomotor ataxia suffer from pain on account of which they have to take enormous doses of morphine. This habit is soon acquired, but notwithstanding the dose of the drug paroxysmal pain continues. Its severity simulates the crises of the primary disease. It becomes a very difficult matter, often impossible, to decide whether the pain is due to the morphine habit or to the primary affection.

Pain in Special Regions. (For head pain, see Nervous Diseases; for pain in the thorax and abdomen, see the respective sections.)

Pain in the Extremities. We have referred to pain in the extremities which may be due to disease of the spinal cord, and pain of neurotic or toxæmic origin. When not due to local traumatic, rheumatic or gouty inflammations, pain in the extremities, unilateral or bilateral, is usually due to neuritis of some form. It is recognized by tenderness in the course of the sciatic nerve at its exit from the pelvis, and by increase in the pain when the limb is extended by forced movement. One of its many branches may be affected, exhibiting tenderness in its

course. Such neuritis is usually traumatic (cold), alcoholic, rheumatic, gouty, or syphilitic; the exact cause in each case must be ascertained by the associate phenomena and the exclusion of other causes. We may distinguish the gouty or rheumatic state on account of which the disease depends, by (1) the history of previous attacks of pain or rheumatism in other situations; (2) the family history of rheumatism; (3) the history of exposure which induced the attack; (4) the character of the pains; (5) the occurrence of pain or rheumatic manifestations in other tissues, as myalgia or pain in the fascia; (6) the occurrence of symptoms of lithæmia (which see), and particularly the character of the urine.

Bilateral pains in the extremities are often of central origin.

Fixed pains in the extremity, in contradistinction to the mobile pains of neuritis, are usually situated in the fascia or muscles, or in the bony structure of the part. They may be the result of strain or injury, which must be carefully inquired for. The latter may be the exciting cause only, if it occurs in a person of rheumatic diathesis, the fixed pain at the situation of the injury being due to the general rheumatic state. Fixed traumatic pains are usually accompanied with tenderness on pressure and are aggravated by movement both active and passive, the tenderness on pressure not necessarily being in the nerve trunk. The special pains about the foot which we are called upon to treat and which have their origin in causes independent of the nerves, or of a rheumatic or gouty diathesis, are:

1. *Pain in the articulations due to flat-foot.* This may be in the tarsus or at the metatarsal articulations. It is a common cause of pain in the extremities and may be unilateral or bilateral. The flat-foot from breaking of the arch can readily be recognized; pressure on the sole of the foot may increase the pain.

2. *Pain in the heel.* This is usually of gouty origin, and is a persistent source of complaint in many instances.

3. *Pain in the interosseous spaces between distal ends of the third and fourth metatarsal bones* (Morton's painful affection of the foot). It occurs in people who stand on the feet a great deal, is relieved by a night's rest, increases as the day goes on, and is increased by pressure or by wearing a tight shoe. It is worse in wet and cold weather. Localized pressure at the point on the sole indicated above causes extreme pain.

We cannot leave the extremities without a word regarding pains in the periphery, in the extremities, of distinctly central origin, the forerunner of hemorrhage into the brain. Mitchell has called attention to these pains. They occur suddenly without evidence of local disease; they are located in one of the extremities, usually the leg, are excruciating, and not influenced by position or local applications or pressure. Occurring in a patient with hard arteries and high pulse tension, they should be looked upon with suspicion.

Pains of the Thorax. Painful diseases of the muscles and of the viscera are excluded; pains of reflex origin will be referred to. They are usually seated in the shoulder or the back, and are due to liver or gastric disease. The pain of liver disease is referred to the right shoulder; of ulcer of the stomach, to the interscapular region and the lumbar region, or to

the top of the shoulder, as in a case observed by Wood. Pain behind the sternum is a reflex neurosis from gastric disorder. It may occur in bronchitis. It may also be due to cancer of the mediastinum, to aneurism, or angina. Pain in the sternum or ribs is syphilitic or due to a periostitis or necrosis following typhoid fever, rarely to cancer. Chronic fibrous inflammation of one or more of the attachments of the muscles is of common occurrence. The pain lasts for years. It is persistent, sometimes associated with stiffness, it is increased by movement, and there may be extreme aching in the parts. The pain of vertebral caries transmitted along the course of the nerve has been referred to.

Girdle Pain. This is a peculiar pain or sensation in the trunk due to disease of the spinal cord. It is described as the sensation of a band tightly drawn around the body. It varies from a simple drawing sensation to extreme pain which encircles the trunk. It is situated above the level of the umbilicus. In milder forms it is due to chronic myelitis or spinal sclerosis; in severe forms, to inflammation of the nerve roots, or to cancerous, syphilitic, or tubercular disease of the meninges.

Pain in the Spine. Pain in the spine is due to organic disease of the cord, to acute or chronic inflammation of the meninges, disease of the bones of the vertebral column, or to curvature of various forms from muscle weakness. Rhachialgia and tenderness in the course of the spine occur after concussion. In organic disease of the cord pain is referred to the loins, the sacrum, or the parts about the spine, but not to the spinal column itself. In the same disease of the cord we have the eccentric or radiating pains of which mention has been previously made, due to irritation of posterior nerve roots. They may be dull, resembling those of rheumatism. In acute cases the pains are accompanied with febrile symptoms which may simulate rheumatism, especially when other spinal symptoms are in abeyance. In chronic cases these peripheral spinal pains are influenced by the weather, which likewise makes it difficult to distinguish them from rheumatism. Rheumatic pains in the limbs independent of actual joint changes occurring particularly after middle life, should lead to examination for loss of power of walking properly, ataxia, and alterations in reflex action, by which their true origin may be recognized. In locomotor ataxia sharp and darting pains occur, "pain crises," and girdle sensations.

Fixed localized pain at some point in the vertebræ when not due to other conditions points to local caries, or may be of syphilitic or tubercular origin, or due to pressure, as by an aneurism.

Pain due to vertebral disease is both local and radiating, it is increased by pressure directly on the spinal column (on the head), by heat or cold, or by electricity applied over the part. It is relieved by removing the pressure of the weight above, as by raising the head or shoulders. It is increased by pressure or a jar on the head. It is relieved by the absolutely recumbent posture. The movements (flexibility) of the spine are interfered with; there is local spasm of muscles; there may be deformity. When the patient is placed upon a flat surface the normal lumbar arch is changed. The pain of curvature from muscular weakness extends

along nerves, is afebrile, without signs of organic disease above mentioned, but with muscle weakness, and general signs of debility. Pain due to *meningeal disease* is local and radiating. It is associated with muscular spasm and stiffness of the spinal column.

Pain in the Side. One of the most frequent sources of pain of which complaint is made to the practitioner is pain in the left side—the so-called infra-mammary pain. By a discussion of it we can show how pain, as a symptom, must be investigated in order to determine the tissue affected and the nature of the disease. The tests used in the study of nerve affections (*q. v.*) are not given. It may be due to many causes, to exclude any one of which inquiry as to the mode of onset, duration, and character of the pain must be made. The structures underneath and about the seat of pain must be examined. 1. The *skin:* to exclude any swelling or tumor or herpes zoster, and to determine the tender nerve points. 2. The *muscle:* to exclude myalgia or pleurodynia. Examine for tenderness; note the effect of movement; does full breathing increase the pain? Palpate with the fingers and the whole hand. Negative answers exclude any muscular affection. 3. The *nerves.* (*a*) Tender points; (*b*) herpes; (*c*) other vasomotor appearances. 4. The *pleura.* Auscultate for friction, if pleuritis. Inquire for cough. Note the character and effect of breathing. 5. The *pericardium.* Note friction of pericarditis or thrill by palpation. Is the heart disturbed in function? 6. The *heart.* It is rare that disease of this organ causes pain, although it may be present in dilatation. Is it affected in a reflex manner, causing palpitation or irregularity? Look for distant disease. 7. The *stomach.* A dilated stomach may, by pressure upward, or gastric disorder in a reflex manner, cause pain.[1] 8. Examine the *vertebra* for disease of it, or pressure upon it by aneurism. 9. The *blood,* for anæmia. 10. *Toxæmia.* Inquire for its presence; particularly malaria, rheumatism, lead, etc. If a local cause is not ascertained look for central nervous or reflex disorder. The above course must be pursued in this, and should be pursued in all cases of pain.

Although any one of the above conditions may cause pain in the side, it is usually—1, a reflex pain from gastric disorder; 2, pain from neuritis; 3, a true neuralgia, from anæmia; 4, a neuralgia from heart fatigue. (Hilton.)

It is to be observed that every local tissue must be examined, and questions asked as to the various attributes of the pain.

Pain in the Loins. When acute, without fever, it may be due to lumbago, to a sudden uterine retroversion, to a suddenly moved kidney,

[1] Shoulder-tip pain, due to anastomosis of phrenic nerve with 3d and 4th cervical and to parts of liver and round ligament (Hilton); or of phrenic nerve and subclavius (Rolleston); or of vagus with spinal accessory, which communicates with 3d and 4th cervical. The v. and s. a. are sensitive to pressure. (Embleton.)

Infra-mammary pain (6th, 7th, and 8th intercostal spaces). The aorta at left side, 3d dorsal vertebra, is in relation to the 4th, 5th, and 6th intercostal nerves through the sympathetic ganglia, through which also the heart sympathetics are in anastomosis. The 4th, 5th, and 6th intercostal nerves supply cutaneous branches to the 6th, 7th, and 8th intercostal spaces. The infra-mammary pain is a reflex neuralgia expressive of some heart distress. The latter is brought about by exhaustion of the medullary and vasomotor centres, from worry or overwork, or from long-continued irritation of the uterine nerves. In leucorrhœa this pain is most common. (Jacobson; Hilton on "Rest and Pain.")

or calculus of the kidney; with fever, acute Bright's disease, smallpox, muscular rheumatism, tonsillitis, influenza or spinal meningitis must be looked for.

CHRONIC PAIN IN THE BACK; BACKACHE. This may be due to many causes. The cause varies with the seat of the pain. When in the region of the kidneys, they should be examined. Organic disease (Bright's) may be associated with backache; more frequently pain, if in one kidney, is due to a calculus or to accumulation of uric-acid gravel. It may be constant in moved or movable kidney. When low down, just above or over the sacrum, it is due to disturbance of the pelvic viscera. The uterus, the rectum (impacted, cancerous) must be examined.

Otherwise we may have—(*a*) Pain due to affections of the *muscles.* 1. *Myalgia of rheumatic origin.* Increased by movement, by dampness, by pressure. Often relieved by warmth, by the recumbent posture or rest. It is associated with symptoms of lithæmia and of the passage of red sand in the urine. When the fascia is affected, or the ligaments of the vertebræ, the upright position and pressure in small areas increase the pain; other muscles may be affected alternately. 2. *Myalgia from sprain.* A history of injury is obtained. Usually one side greater than the other. Tenderness is present and movement increases the pain. There may be increased swelling, vasomotor disturbance, or ecchymoses. Neurosis (hysteria) attends the pain. 3. *Myalgia from fatigue.* Not only acute fatigue after exertion, but chronic muscle tire (and nerve tire). The pain is increased on exertion, after mental, physical, or *emotional* effort. The muscles are flabby; the vertebral column is not supported. The patient lounges or supports the back. Deformities are observed. *Neurasthenia, anæmia,* and local exhaustive disease (uterine, gastro-intestinal, etc.) are present.

(*b*) Pain due to affections of the *nerves.* Nerve pain is recognized by the tender points; by vasomotor phenomena.

(*c*) Pain due to disease of the spine, the membranes, or the cord. (See above.)

CHAPTER III.

THE DATA OBTAINED BY OBSERVATION.

The objective symptoms correspond to phenomena in nature. *Method of procedure* for determination as to patient; *method of the observer*. Inspection, palpation, percussion. The instruments required. Examination of the *exterior*. General examination. The first-sight impression. *The temperament and constitution. Apparent age. Effects of habits and occupation. The attitude and gait. General form and nutrition.* Changes in size and weight. From the amount of adipose tissue—obesity—emaciation. Changes in the skeleton. Enlargement — acromegaly — osteitis deformans — pulmonary osteo-arthropathy. Diminution—rhachitis—osteomalacia. *The exterior in general.* The *skin.* The color—redness—pallor—jaundice—cyanosis—the bronzed skin—Addison's disease — chloasma — tinea versicolor—vagabond's disease—argyria—freckles. The nutrition. Moisture and dryness—hyperidrosis—anhidrosis. Scars. Hemorrhages—mode of recognition—cause—significance. Eruptions—their clinical significance—nature of the lesion—distribution—associate morbid phenomena—general symptoms. Table of skin diseases. Erythema—herpes—erythema nodosum—urticaria—medicinal rashes—erythema of infectious diseases—roseola —miliaria or sudamina. General diagnosis. *The subcutaneous connective tissue.* Œdema—causes—mode of recognition—situation—feet, face, arms and head—œdema of trichinosis—angio-neurotic œdema. Myxœdema. Connective tissue dystrophies. Scleroderma. Sarcomata—cysticercus cellulosæ—brawny induration. Subcutaneous nodules. *The temperature. Fever.* Causes of body heat and fever — mode of determination — physiological variations — pathological variations—the types of fever—the course of the fever—initial stage, fastigium, defervescence—crisis—lysis—the daily range—recrudescence. The symptoms of fever. Subnormal temperature. The diagnostic significance of fever. *The general musculature. General abnormal vital conditions.* Fits or seizures—coma — collapse — shock. *Local examination of the exterior.* The *face*—the facial expression — the *head*—facial hemiatrophy — the *hair*—the *cranium.* Hydrocephalus. The *lips.* The *eye.* The *ear. The neck*—the thyroid gland, the bloodvessels of the neck. *The extremities—hands*, skin, progressive muscular atrophy—lead-poisoning, rheumatoid arthritis, athetosis. *Fingers.* Heberden's nodosities—contraction of fascia—Dupuytren's contraction—deviations in shape. The *nails.* Tropho-neuroses — cold hands and feet — Raynaud's disease — erythromelalgia. *The lymphatic glands. The muscles.* Myositis—idiopathic muscular atrophy—pseudo-hypertrophy—Thomsen's disease—paramyoclonus multiplex. *The bones.* Nodes. They are the physical expression of present disease, or of the ravages of past affections. *The joints*—synovitis—rheumatism—gout—rheumatoid arthritis—the tabetic joint—the hysterical joint—special joint affections. Diagnosis.

The Objective Symptoms. The objective symptoms of disease are the most important to ascertain. They are the "handwriting on the wall." The impress of forces for good or evil is observed. In deter-

mining them we also determine the condition of the organism; its state after the action of the forces of its environment. The physical and mental status of the being is measured. He is individualized. The objective symptoms are data by which a complete diagnosis is made. Without such data the diagnosis is guesswork—one of probability. With such data alone, if accurately, precisely collected, a positive diagnosis very frequently can be made. A correct diagnosis depends upon the skill and thoroughness of the physician and his ability to interpret the data secured, always provided that clear, succinct data can be obtained.

The data obtained by inquiry, within the limitations previously indicated, if such exist, are recorded and then the data obtainable by observation are to be looked for. A physical examination of the patient must be made, followed by an immediate study, or, if time permits, a study at leisure of the fluids of the body, microscopically, chemically, and bacteriologically. By the physical examination a general survey of the individual, including an estimation of the height and weight, is made, and all the organs interrogated by the senses applicable to the investigation of each, with the special instruments devised for aiding them. The natural secretions and discharges, abnormal discharges, all exudations or transudations, and cystic fluids are passed upon.

The student will soon learn that the process of ascertaining the objective signs of disease is in no respect different from that which obtains in the study of any objects in nature or any life phenomena. The chemist notices the form, the color, the density, etc., of the object under examination; the effects of heat and cold, of various reagents upon its structure; determines its component parts and ascertains its relation to other objects in nature. From data thus derived by the use of all his senses he classifies the object. The biologist not only notes the physical appearance of a given form of life, but also notes the phenomena of the living, sentient matter under all conditions in a varied environment. By comparison and analysis, the living being is classified.

By the same powers of observation and the same analytical process, the departures from health are recognized and classified. Is it not, therefore, a wonderful aid to the diagnostician to have had trained previously, by observation in allied sciences, the faculties which contribute to the development of these powers?

What has been thus imperfectly said is ntended to emphasize the fact that no mystery attends the recognition of the objective signs of disease. Patient training, skill in technique, and opportunities of observing disease at the bedside are essential.

Method of Procedure. The method by which the data ascertained by observation are secured is modified by the circumstances under which the patient is seen. It is obvious that the patient who comes to the office, or is not sufficiently ill to be in bed, has sufficient strength to stand, and should be given an exhaustive examination. Moreover, we can inquire into certain abnormalities, as the gait, not visible in bed. On the other hand, from the bed-patient we learn of the position he assumes when lying down, and have better opportunities for thorough examina-

tion of the various organs. Often the objective examination must be very brief on account of the extreme illness of the patient. It may be advisable, although unfortunate, to exclude one or more methods, as percussion if there is pain, or auscultation if there is great restlessness or orthopnœa.

If a complete examination is made, it is well to begin with the exterior. After the *external examination* is made, the *internal examination* is conducted, by grouping together and examining organs functionally related, as the heart and bloodvessels in diseases of the heart; the nose, larynx and lungs, in diseases of the latter. The student may well begin at the head and take up organs in their continuity.

Comparison. The results attained by observation are based upon comparison; the student must bear this constantly in mind. We *compare* the body as a whole with our conception of the normal individual formed by a study of a large number of persons. We compare symmetrical parts—the right side of the chest with the left, the arm suspected to be the seat of disease with the healthy arm, etc. The cardinal rule in an examination is to base the significance of ascertained facts upon comparison with known normal conditions.

Methods of Observation. Securing the Data. To accomplish these ends, examination is made by the sense of *sight* (*inspection*); by the sense of *touch* (*palpation*); by the sense of *hearing* (*auscultation*); and by the sense of hearing applied to the discrimination of sounds developed by *percussion*. By percussion or tapping the part, we also elicit the peculiar phenomena known as *reflexes*.

The sense of *taste* is not used to determine the objective phenomena of disease. By the sense of *smell* some data are ascertained, as the odor of the exhalations and discharges.

Inspection. By inspection we judge of the physical condition of the whole or a part of the body, as seen in the shape and size and in the color; of the vital condition by the expression of countenance, by the character of the movements of the body as a whole or in part, by the position in bed, and by the gait. The appearance of fluids (blood) and of discharges is also observed. Accuracy of the results of inspection as to size is obtained by the use of scales to secure the *weight*.

In order that the data obtained by inspection may be complete and accurate, every portion of the body, and of its internal cavities which can be seen by the unaided or aided eye, should be inspected. The clothing should be removed and, bearing in mind the proprieties, the whole body should be examined. For this purpose the patient should be under a good light. The light should always fall directly on the surface. The entire surface, of course, need not be exposed at once, and circumstances may be such that only one portion need be examined. Nevertheless, the fact must be insisted upon that patients who have been ill for a considerable time, as well as all grave cases, should be examined all over. It is even more important to do this if the patient is comatose. A node on the tibia, undue prominence of the vertebræ, a special rash about the anus, may teach a lesson

which could not be obtained in any other way. It is assumed that the patient has been examined lying down. In nervous diseases and diseases affecting the muscles and bones, observation should be made of the gait of the patient, his ability to stand, the method of rising or assuming a sitting posture, and the performance of other customary physiological acts. For this purpose, as above intimated, portions of the body can be covered, or a light gown should cover the patient from head to foot.

Method of the observer. In order further to secure the data in full, the student should teach himself a method of observation which shall include all the facts that can be ascertained by inspection collated in regular systematic order. Whether the examination is general or local, whether the whole of the body is referred to or only a part, as for instance the nose, the student should accustom himself to make observations in the following order: First, the shape or contour (expression); second, the size; third, the color; fourth, the movability and the physiological condition of the part on movement. If this plan is pursued, little, if anything, will be overlooked. A similar order in the investigation applies to the estimation of the character of the secretions and excretions of the body.

Inspection of special regions. In the inspection of special regions, in addition to ordinary light, artificial light and special instruments are required. The artificial light should be that which is secured from an Argand burner or from a gas-jet with a reflector, or from electricity. To facilitate the examination, the room should be darkened and head-mirrors used as reflectors. A number of these have been devised, any one of which is suitable if it fits the head well and can be adjusted with comfort so that the observer can throw the light on the part he wishes to examine, and at the same time peer through the centre of the mirror. A special arrangement of the patient and the light is required. The patient should sit in an easy, comfortable, erect position, with the light on a level with the part to be examined, a little behind and to his right or left, according to the convenience of the operator. Special apparatus is required for the examination of each cavity: mirrors, tongue depressor, and specula for the throat, an ophthalmoscope for the eye, etc. (See respective sections.)

Palpation. The results of inspection are always confirmed when possible by palpation, the sense of touch supplying also additional data. The nutrition of the parts is ascertained. The density, the resistance, the special character of the part as indicated by the density, whether solid or liquid, are determined by this method of examination. On examination of the skin, the degree of dryness or moisture; the character of the skin, whether smooth or rough; the density of the part, as to degree of thickness and resistance, are all ascertained by means of the sense of touch. The presence or absence of pitting is observed, and the nature of swellings ascertained. In a similar manner local areas are examined. The same routine method should become habitual with the student. First, the shape and contour; second, the size; third, the character of the color, its change on pressure, etc.; fourth, the movability of the part, and

the character of the normal movements, as when a joint is under observation; fifth, the resistance and density of the part examined, or special characteristics revealed by touch—the elasticity of skin, firmness of muscles, and in swellings the presence or absence of fluctuation. In addition to the above, other phenomena are detected which belong more particularly to the living body. By palpation, alone or with instruments, we determine the sensibility of the part, the presence or absence of tenderness, the temperature and the degree of moisture. In the examination of special regions by means of palpation some phenomena are determined peculiar to the system under examination, and dependent upon its physiological or functional action. Thus in palpation of the chest, in addition to its movement, we note the vibrations transmitted to the hand when the patient is asked to speak, or detect abnormal vibrations from the friction of two rough surfaces together (pleura), or from the throwing of fluids into agitation: fremitus, friction, and râles are thus transmitted.

Knowledge of the action of the heart and of its position is ascertained by palpation; thrills are detected, abnormal impulses felt. (For method of procedure, see respective organs.)

Auscultation. By auscultation, we ascertain and analyze the sounds that attend the physiological performance of such organs as produce sound: the lungs, the heart, and the bloodvessels. Abnormal sounds may be created in the pleura and pericardium and in hollow viscera, as the œsophagus, stomach, and intestines, and their presence is ascertained by auscultation. (For methods, see under Diseases of the Lungs and Heart.) The character of the voice is also studied for abnormalities in the respiratory tract, which are indicated by change in the quality or loudness of speech.

Percussion. By percussion we strike over organs and elicit sounds which indicate the physical condition of the part percussed. In health the lungs and the gastro-intestinal tract contain air in certain definite relations, and therefore the sounds yielded by percussion are always of a definite character. Any change from the normal sound is indicative of disease, of abnormal structure, or of alterations of the normal relations of the parts. Percussion determines these changes, and in addition we are enabled to estimate the size of organs. It is possible to determine the size of the liver, the heart, or the spleen, because of the relationship of these airless, non-resonant bodies to the air-containing structures around them. As this method of securing data is of greater use in pulmonary and abdominal diseases, the mode of procedure will be described under chapters upon diseases of the lungs and abdomen.

In addition to the data obtained by the above methods, valuable and essential data are obtained by chemical, microscopical, and bacteriological examinations of the fluids, discharges, exudations and transudations, and by aspiration and special examination of the fluids obtained from the natural cavities or from cysts of the body. Bacteriological diagnosis and exploratory puncture will be considered in a special chapter.

The Armamentarium. The following instruments are necessary to conduct ordinary methods of investigation:

To aid the eye, we have the microscope; various reflectors and mirrors to illuminate cavities, as the ophthalmoscope, the laryngoscope, and the otoscope; specula of various kinds, and forms of illumination, as the Argand burner or electric light.

To aid the touch or confirm its findings, the thermometer, the tape-measure, the cyrtometer, the dynamometer; the plessor to ascertain reflexes; the æsthesiometer to determine the keenness of sensation; sounds for the œsophagus; probes for the nares; the sphygmograph for the pulse.

To ascertain the nature of the contents of a swelling or tumor, or of the natural cavities of the body, the exploring needle and aspirator are used. The contents of the stomach, the bowels or the bladder must be obtained often, and for this we use tubes or catheters, the fluid being withdrawn by suction or by siphonage.

The sounds that are elicited in order that the sense of hearing may be utilized are evoked by the use of a plessor and pleximeter, and are localized and differentiated by the stethoscope, of which there are many varieties.

For the examination of the blood, special instruments are employed—hæmocytometer and hæmoglobinometer; for the urine and other fluids, chemicals, specific gravity bottles, etc.; and for bacteriological research, the various appliances that appertain to such investigations. The instruments above mentioned will be detailed in the respective sections, and their method of employment indicated.

The Microscope. This instrument is employed for the investigation of the phenomena of disease in nearly all the organs or tissues. It is absolutely essential for clinical work. It need not be described. It is enough to say that lenses which amplify from 50 to 1500 diameters should be secured, an oil-immersion objective, and an Abbe condenser. Low powers are necessary for the study of plate cultures, and for the inspection of comparatively large objects in the urine, sputum and fæces. High powers are necessary for bacteriological work. The diaphragms must be used with the Abbe condenser.

Data obtained by Examination of the Exterior.

The examinations are made by inspection and palpation (see above). All clothing should be removed and the examination made in a good light. Comparisons of the two sides of the body should always be made. The examination is both general and local.

External changes due to or associated with disease of special systems are considered under the examination of the system concerned, as the bony surface and bones (contour) of the thorax in the examination of the respiratory system, the abdomen inthe examination of the digestive system.

A. General Examination of the Exterior.

The general appearance of the patient affords an idea of the ability with which he has been able to cope with the antagonistic forces of his

environment, or to overcome the deleterious effects of his occupation, or indicates the effects of present or past disease or of disease derived by heredity. The *first sight*, striking impression, is always to be noted. "Very sick," "comatose," "collapsed," etc., or "robust," "cyanosed," etc., are speaking memoranda. To the experienced practitioner, the opinion formed by the first glance is often of great diagnostic significance.

We then note—*first*, the temperament and constitution of the patient or the evidence of any diathesis or cachexia; *second*, the apparent age; *third*, the indications, from his appearance, of the habits and occupation of the patient; *fourth*, the position assumed in standing, walking, or in bed; *fifth*, the general form and nutrition; *sixth*, the occurrence of fits, coma, collapse, or shock.

A general review of the exterior will often indicate the probable system that is the seat of disease. For instance, violent respiratory action points to the lungs, paralysis to the nervous system, the enlarged abdomen to disease of viscera of that region.

1. **The Temperament and Constitution of the Patient.** In former times emphasis was laid upon general appearances as indicative of a particular diathesis, or inherited constitution of the patient. Five varieties of diathesis were described to which general appearances pointed. They were the gouty or sanguine arthritic, the strumous, the nervous, the bilious, and the lymphatic diatheses. While certain appearances point to the occurrence of groups of individuals who may be classified under one of these diatheses, it is well not to lay too much stress upon them for diagnostic purposes. Certainly, as pointed out by Gairdner, it is not proper, after a survey of the patient, to note the presence of any particular diathesis in so many words—as the lymphatic diathesis. The student should teach himself to note individual appearances, and after a complete examination is made or the light of experience supports him, draw a final conclusion as to the diathesis.

In the gouty or sanguine diathesis, the osseous system and muscles are well developed, the nutrition active, and the patient usually robust in appearance. The digestion is good, respirations deep, the circulation is well carried on, as shown by the florid skin and the large heart; the pulse is firm and steady, and the pressure in the arteries is high. The head is large and the jaw prominent, the teeth good. The hair is strong and thick. The individual with such diathesis is predisposed to the arterial changes of advancing age. Apoplexy, aneurism, and angina pectoris, or resulting complications of the senile changes in the heart and arteries, develop.

In the strumous diathesis the appearance of bones, the glandular system, and the face are expressive; the bones of the chest are small; the long bones are slender, while their epiphyses are large; the forehead is broad and prominent, the lips full, the alæ nasi thick, the teeth are carious, the lower jaw light and thin, the hair is fine and often of a light hue, the eyelashes long, the eyebrows arched, often heavy. In this diathesis the nutritive changes are poor, inflammations are usually sluggish; disease of the bones, of the glands, and forms of tuberculosis, are liable to be more severe.

In the nervous diathesis we see small, active, restless beings, with small bones and large muscles. They are full of energy, and carry on large business or mental operations. The features are well formed, the eye active. They are the subjects of overwork and early breaking-down of the nervous system, and of dyspepsia. They possess idiosyncrasies toward drugs, as opiates.

In persons of the bilious diathesis, we find a dark skin, dark hair, muddy conjunctivæ. They are usually not well nourished. Their digestion is poor, and they are subject to so-called attacks of biliousness. Sick headaches are common. Fatigue is not borne well.

In the lymphatic diathesis there is lack of energy and sluggishness of nutritive processes ; such persons are unable to keep up in the wear and tear of life. They are usually pallid and have soft muscles.

In addition to diatheses, *cachexiæ* are also noted. Cachexiæ arise from the ravages of disease and especially from such forms of disease as cause reduction in the number of the red cells of the blood and diminution of the hæmoglobin. The cachexiæ that have been described are caused especially by syphilis, gout, and chronic malarial poisoning ; in cancer of some part of the digestive apparatus—and, indeed, in all forms of chronic disease of the digestive tract—a cachexia is seen. The anæmia that arises from lead, arsenic, and other metallic poisons produces an appearance to which the term cachexia has been applied. The special cachexia derives its name from its cause, as the syphilitic or cancerous cachexia.

2. The Apparent Age of the patient should be estimated from his appearance and compared with the *exact* age when this is learned later. In this way the physician will be enabled to judge whether the patient is aging too rapidly or bearing his age well. An obvious advantage from noting the age of the patient arises from the fact that it enables us at once to exclude a large number of diseases which are not found in the period of life to which the patient belongs. For example, if the patient is a child we have not to consider the chronic degenerations and the visceral cirrhoses which appear in middle and later life. Conversely, in an old person we do not expect to meet with the exanthemata, which affect children almost exclusively. So, too, typhoid fever and consumption are more common in adolescence and early manhood than in childhood and old age. Again, in very young girls, the question of menstruation and its difficulties never have to be considered. *Gray hair* in a person under thirty-five generally indicates a feeble constitution and premature age. *Loss of hair* is not significant, for, apart from a tendency to baldness which is very marked in some families, professional men who do much brain-work, especially in hot, close rooms, are apt to become bald much sooner than other men. The presence of *wrinkles* at the corners of the eyes and of "crow's feet," and of dull, dry, lustreless eyebrows, should be noted as indicating aging, whether the person has lived long or not. In women approaching forty who do not gain in flesh there is often a suggestive prominence of the angles of the jaw and sterno-mastoid muscles with a certain loss of roundness and elasticity of the cheeks. The latter appearance, however, may be due to loss of molar teeth.

3. Effects of Habits and Occupation. From the general appearance, the habits of the patient as to industry, neatness, or care of dress, may be observed ; they are of diagnostic importance, particularly in brain affections. The appearance also shows frequently whether the patient is addicted to alcoholism or the use of other narcotics.

The occupation of the patient is often important in throwing light upon his disease ; the brown, weather-beaten face of the farm laborer, sailor, or driver contrasts strongly with that of the merchant, clergyman, or clerk. A machinist can often be recognized by his grimy, oily hands. All this information can be obtained by a glance, and many details can be added before the patient has taken his seat in the consulting-room.

4. The Attitude and Gait of the Patient. The attitude of the patient gives information as to his physical vigor, and, to a certain extent, of his alertness of mind. A man vigorous of mind and body will stand firmly upon both feet, with back straight and shoulders square, and head erect. When one is depressed by care or disease the shoulders have a tendency to droop, and the head to fall forward. Indecision and vacillating disposition are sometimes indicated by the patient standing first upon one foot and then upon the other while talking, or by an unsteady look from the eye.

When one shoulder is lower than the other and the patient is of phthisical build, pale and emaciated, the attitude is strongly suggestive of phthisis or chronic pleurisy of the side on which the shoulder is depressed.

Sometimes in acute pleurisy the patient will walk with the shoulder depressed and the arm firmly pressed against the affected side so as to restrict its movements as much as possible.

Decubitus. The attitude of the patient in bed is often significant. He may assume the active dorsal or side position, with the body arranged so that it is comfortable and unconstrained. Then slight indisposition only is present. On the other hand, the side position, the dorsal position, or the upright or semi-upright position, may be assumed.

To the close observer the attitude of a patient in bed is sometimes reassuring. He lies easily upon his back or turned slightly to one side with the arms uncovered, and he may even turn or sit up to meet the physician as he enters the room—each of which point to moderate illness or to the onset of convalescence.

Side Position. A patient with acute pleurisy or pneumonia will lie on the affected side so as to limit its motion as much as possible. The breathing will be shallow and frequent, the expression of the face anxious, and occasionally a spasm of pain contracts it as the patient coughs or is obliged to take a full breath. He usually lies on the affected side because fixation is thus secured and pain on inspiratory movement diminished, and also because there is greater liberty for expansion of the free healthy side. If effusions are present, by lying on the side of the effusion pressure is removed from the heart and the unaffected lung, an obvious point of advantage.

At times in cases of thoracic aneurism, if situated on one side, or of

movable thoracic tumors, the patient will lie on the side which is the seat of the disease.

The *dorsal* position assumed in health or slight disease has been referred to. When the position is assumed in grave disease the term *passive* is applied to it because it is often assumed without the volition of the patient.

In grave cases of typhoid or other low fevers, the patient lies upon the back and shows a marked tendency to slip down in the bed. The expression of the face is heavy or vacant. The lips and teeth require constant cleaning to keep them from sordes; the tongue is dry and glazed or covered with sordes; the tendons of the wrists twitch convulsively, and the patient lies with open or half-open eyes (coma vigil), picking at the bedclothes or at imaginary objects which float before his eyes.

A healthy baby a few months old finds motion an almost ceaseless delight. It will lie on its back, kick up its feet, play with its toes or some object that attracts it, crowing, wriggling, squirming. In *rickets*, on the contrary, the little patient lies as quiet as possible, even refraining from crying because all motion is painful. In cerebro-spinal *meningitis* the head is drawn backward and downward and the muscles at the back of the neck are rigidly contracted.

In *acute disease* involving the peritoneum or neighboring organs, such as acute peritonitis, appendicitis, or endometritis, the patient lies on the back with the legs flexed upon the thighs and the thighs upon the abdomen. Motion is avoided as much as possible, and so is any pressure upon the abdomen.

Lying in the *lateral* or *dorsal* position, with legs drawn up and trunk and head drawn down to meet them with groans of pain and possibly involuntary bearing down, is seen in hepatic and intestinal colic, and is suspicious of the throes of labor, if the patient is a woman.

The Semi-upright or Upright Sitting Position. In an acute attack of *asthma* the patient is found sitting up in bed, or in a chair, possibly by an open window. The expression of the face is anxious, the skin dusky or pale and moist. The breathing is loud, noisy and scraping. The demand for oxygen is imperative, difficulty is experenced in inspiration and expiration, not enough air being able to enter the alveoli for physiologic purposes; expiration is prolonged and labored (expiratory dyspnœa). The patient sits with the chin raised and head erect, the hands grasping the arms of a chair or the bedclothing, so that by fixing the chest the accessory muscles of respiration can be of the greatest assistance in supplementing the diaphragm. In emphysema, in its late stages or complicated with bronchitis and asthma, the same position is assumed almost constantly.

In *pericarditis* with effusion, in *large pleural effusions*, and in *advanced heart disease* with anasarca the patient is unable to lie down on account of the smothering feeling which the recumbent position induces. In pericarditis the expression of the face is extremely anxious, the patient having a dread of impending death.

In large pleural effusion the expression is not usually so anxious, but the dyspnœa may be intense. The patient is propped up in bed, leaning

slightly to the affected side, and devotes all his energies to breathing, avoiding any exertions such as moving, answering questions or coughing, which tax his breathing muscles still more. One side of his chest may be observed to move violently while the other is motionless.

In heart disease and anasarca, dyspnœa frequently amounts to *orthopnœa*. The patient may be found propped up in bed or seated in a large rocking-chair, some patients finding greater comfort in the latter. The face is pale, livid, or jaundiced, and may be swollen, while the cellular tissue throughout the body is œdematous and the cavities, especially the peritoneum, are more or less filled with fluid. In diaphragmatic pleurisy the position assumed is very characteristic—the erect sitting posture, with the body leaning forward and laterally, to relieve the pain.

The Prone Position. Rarely the patient is found lying upon the abdomen. When seen in this position it is because of relief to abdominal pain, or to colic of any form, or from relief that is given to an ulcer of the stomach, and aneurism, or caries of the vertebræ on account of the position.

In tetanus, muscles are in a state of tonic contraction, on account of which an unusual position is seen known as *opisthotonos*, in which the body rests on the head and heels, the trunk being arched upward. In strychnine poisoning with tonic convulsions the same position may be assumed.

Emprosthotonos, vaulted side position, is occasionally assumed in tetanus and also in strychnine poisoning.

Unclassified Positions. Irregular or bizarre positions are usually assumed in affections of the nervous system, particularly in hysteria.

Restlessness. Often the patient is unable to assume a position, or at least to remain fixed in any position. This may occur on account of pain, or because of irritation of the nerve centres previously described. In cases of moderate cerebral hemorrhage, and of shock, there is great restlessness. The patient is restless without the appearance of agitation. In profuse hemorrhage, whether uterine, intestinal, or pulmonary, on account of cerebral anæmia, there is also restlessness with sighing and gasping. The pallor that attends the hemorrhage, the quicker pulse, the great thirst, with the history of bleeding, are sufficient to explain the restless state. In *chorea* there is more than restlessness, there is constant twitching of muscles with jerking from one side of the body to the other. The patient does not keep the covers on and in her agitation often does herself considerable injury.

In *cerebral meningitis* the patient tosses from side to side, or lies with the head retracted and pressed deeply into the pillow. The eyes are injected, the pupils contracted, and frequent sharp cries are uttered, especially if the patient be a child.

In *hysterical convulsions* the patient, usually a young woman, tosses wildly to and fro, screaming, laughing, or crying; or coma may be mimicked. The moods change often with great suddenness. The appearance is very alarming at first sight; but the pulse and breathing are not much accelerated, there is no fever, and the patient is conscious enough not to injure herself even to biting the tongue.

THE GAIT AND STATION. Both are of great significance, particularly in the study of nervous diseases. The terms astasia and abasia are applied to the loss of power of standing and of walking respectively, without paralysis. Both are usually functional or hysterical.

The gait is sometimes characteristic. The *hemiplegic* patient advances the sound limb, and then brings the other up to it by lifting the pelvis and swinging the paralyzed limb round by a movement of circumduction. The shoe is worn down at the toe in an irregular way. Sometimes the shoulder on the sound side is thrown outward and forward so as to facilitate the raising of the pelvis on the paralyzed side in order that the limb may be circumducted. The arm may be rigid or bent at the elbow, the fingers being flexed upon the palm, and the thumb turned in.

In *locomotor ataxia* there is uncertainty in the gait, which may only be felt by the patient or be apparent to the observer also. There is irregularity in the line of progression, or the movements become very jerky and erratic. As there is very little motion at the knee, because it is spasmodically braced, the pelvis is slightly tilted until the foot is released ; the foot is then raised unnecessarily high, jerked rapidly forward and outward and brought down with a sudden stamp, or flail-like action, on the heel. The patient's centre of gravity undergoes several changes at each step, so that he swings from side to side. He cannot walk in the dark, and at a later stage requires the aid of canes to prevent him from falling forward.

In *paralysis agitans* the attitude and gait of the patient are peculiar. The head and body are thrown forward and fixed in that position ; the arms are slightly abducted and partly flexed, the hands being in the position in which a pen is held. The legs are also bent at the knees. Rhythmical tremors affect the hands first, and then the rest of the body, the head and neck usually escaping. On attempting to walk, the gait is *festinating*, that is to say, each step becomes more rapid than the preceding, until the patient is prevented from falling only by catching something. The tremors cease during sleep, and are independent of voluntary motion.

In *spastic paraplegia* the patient walks with two sticks. He leans on the left one, arches the back, and then lifts the pelvis and the right limb as far from the ground as possible, but cannot quite clear it. The toe has a marked tendency to stick to the ground, and is brought forward with a scraping sound. The knees have a tendency to interlock, and the foot which is brought forward is apt to cross in front of the other.

In *disseminated insular sclerosis* the gait is somewhat jerky and resembles the gait of ataxia, or of tumor of the cerebellum. Of course the recognition of the disease that causes such peculiarity in gait cannot be made without observation of the mental and nervous phenomena that attend such affections. In *hysterical paraplegia* there is sometimes complete loss of power of standing or of walking. The patient will fall if an attempt is made to compel her to stand. Or she will walk with the knees and the hips semi-flexed, or in awkward attitudes, implying muscular effort greater than that needed for the normal gait.

It is recognized by the fact of its occurrence in young subjects in whom are observed other striking phenomena of hysteria.

Cross-legged progression. This form of gait is seen in children with spastic paraplegia, and occurs because of contracture in the calf muscles. When the child begins to walk, one foot gets over in front of the other, or swinging oscillation of the body occurs, which may persist through adult life.

The gait of *pseudo-hypertrophic muscular paralysis* is known as the waddling gait. This oscillating character is assumed in order that the body be so inclined "as to bring the centre of gravity over each foot on which the patient successively throws his weight, because the weak gluteus medius cannot counteract the inclination toward the leg that is off the ground, unless the balance is exact." (Gowers.)

The position assumed in getting up from the floor, as described by Gowers, is pathognomonic. The patient turns over in the all-fours position, raises the trunk with his arms, rests the trunk upon the extended hands, then extends the knees, pushes back with the hands until he can grasp one knee with the corresponding hand, then grasps the other knee and pushes up the trunk by gradually raising the point of support for the hand upon the thigh. The gait of *paramyoclonus multiplex* and of *Thomsen's disease* is also peculiar. (See Muscles.)

Station. Ataxic astasia in *locomotor ataxia.* The inability to stand is observed under many circumstances. Either with (1) the eyes closed, or (2) the eyes open and the toes and heels in contact, or (3) with the eyes open and feet apart. The latter occurs in the highest degree of ataxia and may be followed later by complete loss of power of standing.

Swaying. If a healthy person stands with the eyes shut the body will sway slightly. In a patient with locomotor ataxia swaying is seen in increased degree.

In *pseudo-hypertrophic paralyis,* if the patient stands, lordosis is seen. It disappears entirely when the pelvis is supported, which occurs when the sitting posture is assumed. In the later stages of this affection there is posterior or lateral convexity of the spine with astasia.

In the paroxysms of *Ménière's disease* the loss of power of standing may be absolute. The patient may be hurled to the ground, and be quite unable to rise or sit up. The nature of the paroxysm is suspected on account of the sudden onset and the complaint of vertigo, along with the ear symptoms that attend this affection.

In disease of the *middle lobe* of the *cerebellum,* swaying from side to side, or in large waves, is observed. The appearance is like that of a drunken person. While the walk is peculiar, the patient can usually sit up.

5. General Form and Nutrition. The general form and nutrition of the body are estimated by the color of the skin, the amount of subcutaneous fat, the degree of muscularity, the size and shape of the osseous system. In other words, the degree of robustness is ascertained by the color and the size and shape, including the weight, of the individual. From the above estimation we ascertain the degree of development of the individual. To recognize lack of development is often to be able

to explain phenomena of a functional nature which otherwise could not be done. The color will be considered under the head of the condition of the skin.

Importance of such observation. It is extremely important that these observations should be made, particularly in childhood and adolescence. Not only are marked departures from the normal significant, but slight deviations point to the occurrence of processes which modify nutrition. It frequently happens that it is impossible to explain the occurrence of some functional disorder, as neuralgia, or of derangement of the viscera, or of indefinable ill health, in which the patient has inaptitude for exertion or inability to conduct the usual affairs of life. The recognition of malnutrition as shown in lack of tone of muscles, or diminution of weight, is often sufficient to point the way to successful treatment by general methods.

The Size and Weight. Change in size may be general or local. General increase or diminution in size is due to enlargement or diminution of the bones, muscles, and fat singly or combined. The term *emaciation* is applied to *atrophy* of fat and muscles when it is in excess. If it is accompanied by a great loss of strength the term *marasmus* is employed. When large accumulations of fat take place the word *obesity* is applied to the condition. The estimation of the proportionate size of the patient to his weight is usually based upon the amount of subcutaneous fat. The general accumulation can readily be recognized by rotundity of the exterior. Variations in size, however, may in addition be due to changes in (1) the skeleton, (2) muscles, or (3) adipose tissue, or (4) to accumulations of serum, or (5) abnormal tissue, as mucin underneath the skin, or (6) from connective tissue dystrophies in the same region. Consideration of the latter causes will be postponed; that which here follows refers to the amount of fat and to a certain extent of the muscularity.

Their size affords some information as to the degree of development of our patients and as to the class of disease to which they are most liable. While there is no absolute standard by which to compare the relative proportion of height to girth in individual cases, yet there is a type generally recognized as being usual, and variations from it give rise to such expressions as *stout*, *spare*, *slender*, *thin*, *tall*, and *short*. *Stout* usually expresses an increase in girth and a moderate excess of flesh over the normal. When used in this sense it becomes synonymous with lusty, and indicates an increase of flesh which is well distributed and due to a healthy, active nutrition without impairment of physical activity. In some cases, especially in women, stoutness is used as a euphemism for corpulency, but not often for that excess of fat properly called obesity. Stoutness in the sense of lustiness up to middle life is an indication of physical and often of mental vigor. It is often found in gouty and rheumatic subjects. A tendency to take on flesh after the age of forty-five, especially if the person's occupation is sedentary and his habit of body inactive, is not to be regarded as favorable. It may be compared to a warrior's persisting in wearing an increasingly heavy weight of armor after the campaign is over. Increased weight under such circumstances is not increased strength, but increased burden, and

the burden becomes greater with advancing years. Those who are stout in the sense of having too much fat in proportion to bone and muscle, bear fevers and exhausting diseases poorly under forty. Women at the menopause are very prone to take on flesh rapidly. Fat subjects after middle life, and to an increasing degree after that period, are liable to fatty degeneration of the heart, bloodvessels, and important viscera.

Persons who are tall and thin, especially if they have become tall rapidly after puberty, are commonly looked upon as delicate, and as especially liable to consumption. There is reason for this view. But if they live to be twenty-five or more without disease of the lungs or pleura they may then live to a great age.

Some patients have an appearance which is well described and understood by the word "*spare.*" The form is compactly put together, but with small bones and a scanty allowance of fat. There is a tendency to leanness rather than to roundness of form.

In still others, muscle and bone predominate, and the form is apt to be angular, such as those described as *wiry*. They are often possessed of great muscular power and resistance to strain. Those of spare and wiry habit bear disease very well. Inspection alone may leave one in doubt whether to regard an individual as thin and delicate or spare. Light will be obtained from the patient's occupation and the amount of physical exertion of which he is capable, and also from the tonicity and hardness of his muscles. If one stops to think a moment he will see that for the same amount of heart and lung capacity a man will be better off if spare than if corpulent; because in the latter case he has an additional load to carry, and has to nourish and keep up a thick blanket of fat from which he derives no adequate advantage. Hence a person of spare build who survives childhood and adolescence without disease probably has on the whole a better prospect for long life than a stout person.

Normal Habit. In estimating the size or weight of the patient it is important to ascertain the customary habit of the individual as to the taking on of flesh, and if it developed suddenly or followed acute disease.

Weight. Nothing has yet been said of the weight, but as it affords a precise estimation of the size, particularly if considered in relation to the height and age, the following discussion will include the two points, size and weight.

While the weight of the body can be estimated approximately by the eye and the degree of emaciation noted, the habit should be formed of accurately estimating it by means of the scales. Machines are now made which can be used for weighing the patient and at the same time noting the exact height. It is particularly important to note the weight from time to time. In the course of wasting disease we learn the effects of treatment thereby, or, on the other hand, the march of disease in spite of treatment. In obscure cases, as of tuberculosis, persistent loss of flesh is a serious diagnostic and prognostic symptom. After acute disease, if the patient is weighed every week, the onset of insidious sequelæ, as tuberculosis, may be detected.

The relation of body weight to height is of importance. It is also

important to have knowledge of the average weight of the individual in different periods of life. The progressive increase in weight which should take place after birth should be remembered, as the opposite is positive evidence of malnutrition.

The table of Mr. Hutchinson is sufficient to enable us to judge the average weight of a man of a given height in health:

A man of	4	ft.	6	in.	to	5	ft.	0	in.	ought to weigh about	92.26	lbs.
"	"	5	"	0	"	5	"	1	"	" "	115.52	"
"	"	5	"	2	"	5	"	3	"	" "	127.86	"
"	"	5	"	4	"	5	"	5	"	" "	139.17	"
"	"	5	"	6	"	5	"	7	"	" "	144.29	"
"	"	5	"	8	"	5	"	9	"	" "	157.76	"
"	"	5	"	10	"	5	"	11	"	" "	170.86	"
"	"	5	"	11	"	6	"	0	"	" "	177.25	"

In some life-insurance tables of this country the average weight for the height is lower, especially in persons over five feet ten inches.

Local Weight. It is not to be forgotten that accumulations of fat may take place in special portions of the body; the abdomen is the favorite seat for excessive accumulation, particularly in women and in men of sedentary life with habits of excessive indulgence in food and drink.

Weight in Disease. The question of *weight* is an important one in disease. As has been stated, persons with an excess of fat do not bear fevers and exhausting processes so well as those who have a larger proportion of firm muscles in proportion to their weight. Remember, if emaciation is present, to ascertain its amount and degree, its relation to unusual mental care, or to acute disease. Slow progressive emaciation is of serious moment, as evidence of tuberculosis or disorder of assimilation. Remember the wasting that is associated with great hunger, excessive thirst, and polyuria in diabetes. On the other hand, such symptoms as occasional cough, slight evening fever and impairment of resonance at one apex of the lung, become much more significant of incipient phthisis if accompanied by loss of weight. And at any stage of phthisis a maintenance of the body weight is one of the most favorable elements in prognosis.

Again, while loss of weight attends all the diseases of the digestive tract which interfere seriously with nutrition, it progresses more rapidly and steadily and attains a greater degree in malignant disease than in the mechanical or functional diseases. Hence the question of loss of weight is important in deciding between chronic catarrhal gastritis and gastric carcinoma. But still more important is the question of the time during which loss of flesh has been taking place, and whether it has been progressive or interrupted by periods of gain in weight. If during two or three years the patient has been vomiting occasionally and losing flesh, but gaining again from time to time, it is much more significant of gastric catarrh than of gastric cancer.

False Increase of Weight. In certain cases of great anasarca and in malignant disease of the abdomen, especially huge cysts of the ovary in women, and sarcoma of the kidney in children, there may be actual increase of weight due to the accumulation of water or to the new growth, though the rest of the body is manifestly emaciated.

Weight in Children. In babies and children fat is more likely to be

a sign of good health than in adults. Nevertheless, the quality of the flesh is to be taken into consideration. There are fat and flabby babies and children, and there are others who are fat but whose flesh has a firm, solid feel. The former often gain and lose flesh rapidly, and when ill do not appear to have much resisting power. The size of a child gives a good idea of its nutrition. A child may have its growth stunted by bad food and unfavorable hygienic conditions, or the stunting may be the result of exhausting disease, such as whooping-cough.

Degree of loss. The whole body may exhibit considerable loss of flesh, the cheek bones and temporal fossæ being distinctly visible, the muscles soft, the limbs wasted, and the subcutaneous fat diminished. It is important to notice whether flesh has been lost or not, and how much, and how long a time the loss has been going on. Such facts furnish the clue, not only to diagnosis but to treatment also. Flesh is lost in almost all the acute and chronic diseases, but it becomes of special moment in diagnosis in the latter. It is most noticeable in tuberculosis, cancer, marasmus, cirrhosis of liver and kidneys, diabetes, in anæmias, and in cachectic conditions due to prolonged suppuration or chronic diarrhœa.

Local Change in Size. There may be local increase or diminution in size, alone or combined. When one part is increased in size and another growing progressively small, the incongruity indicates disease (see below). The face is swollen, especially under the eyes and above the jaws, in the dropsy of large white kidney and in parotitis. The neck may be enlarged in the sterno-clavicular notch, or laterally above the clavicles in aneurism. The thyroid as a whole, or both lobes, is enlarged in goitrous affections and in Graves' disease.

The face may be thin and even much emaciated while the abdomen is greatly distended from dropsy or from tumors of the various abdominal viscera or glands. The chest is enlarged or contracted. Local increase in size in thorax or abdomen is significant of tumors.

The head is much increased in size in chronic hydrocephalus, while the face remains small.

The loss in flesh in the extremities or special muscles may be local and atrophic in character, as in some diseases of the nervous system, such as neuritis, infantile palsy, hemiplegia, and monoplegia.

The increase in size may also be local, as in hydrocephalus, elephantiasis, myxœdema, œdema, and various tumors.

Changes in the Skeleton. The degree of development, the size and the strength of the individual in general, may be ascertained by the condition of the bones of the skeleton.

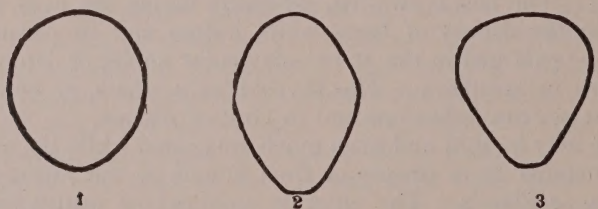
Enlargement of the Bones. In some affections the bones are unduly enlarged, modifying the general form and causing increase in the size of the individual.

Acromegalia. Marie first described *acromegaly*, a remarkable change in the skeleton, in which the bones of the hands, feet, and face are particularly the seat of hypertrophy. The fibro-cartilages of the ear and larynx also enlarge. The enlargement of the inferior maxillary and frontal bones causes the face to assume a peculiar, elongated, elliptical outline. The nasal bones are enlarged, and the nose thickened; the temporal fossæ are deepened on account of enlargement of the malar

bones. The forehead retreats because of the enlargement of the frontal sinuses and projection of the superciliary ridges; the chin is prominent and the lower teeth project beyond the plane of the upper; the lips and eyelids may be thickened; the tongue is enlarged and thickened; the hands present a peculiar appearance; they are much broader, the terminal phalanges are flattened and give the hand a spade-like shape; the nails present longitudinal striations. With the changes in the face and hands there is usually spinal curvature; the abdomen is prominent, and, as before intimated, the height is increased. The muscles become weak and may atrophy; the skin is often pigmented, varicose veins have been observed, and the patient complains of hemorrhoids. The thyroid gland may be atrophied or hypertrophied. It may be well to state in passing that with these appearances nervous phenomena are observed and disorder of special senses complained of. Hemianopsia, limitation of the visual field, and blindness or deafness arise.

Osteitis deformans. Another remarkable change is seen in the skeleton and has been described by Sir James Paget; in this there is marked change in the contour of the patient and a peculiarity in the mode of locomotion. It is known as *osteitis deformans.* The head is advanced and lowered, so that the neck is very short, and the chin, when

FIG. 1.



1 Outline of face in myxœdema.

2 Outline in acromegaly.

3 Outline in osteitis deformans.

the head is at ease, is more than an inch below the top of the sternum. The chest becomes contracted, narrow, flattened laterally, deep from before backward, and the movements of the ribs and spine are lessened; the arms appear unnaturally long, the shafts of the tibia and femur are bent so that the patient becomes bow-legged. There is some stiffness, but no loss of power and not a great deal of pain. The skull is increased considerably in thickness. These changes in the bones cause a dwarfed appearance of the trunk in comparison with the legs and arms, and the posterior lateral curvature necessitates a characteristic attitude. The skeletal changes are noted particularly in the long bones. As a result of the enlargement of the cranial bones, the face presents a triangular outline, with the base above and the apex below (see Fig. 1, outline 3), thus differing in appearance from the outline in acromegalia. (Fig. 1, outline 2.)

Pulmonary osteo-arthropathy. Marie distinguishes acromegaly from another skeletal change in which there is hypertrophy of the bones of the extremities and the shafts. In this form of arthropathy the bones of the head and face are not affected. The hands and feet are enlarged, and the patellæ and other bones of the knee-joints increased in size.

Curvature of the spine is present. The appearance of the fingers is different from those of acromegalia. The ends are enlarged and bulbous and the nails curved in a transverse and longitudinal direction, like the clubbed fingers of phthisis, although the chief enlargement of the fingers is not terminal, and there is no cyanosis as in phthisical clubbing. The change seemed to be associated with pulmonary affections, and Marie applied to it the name *ostéo-arthropathie pneumonique.*

Local changes of the bones are considered in the section on local examination of the exterior.

FIG. 2.

Pulmonary osteo-arthropathy. Female, aged eleven. Tuberculous vertebral caries and pulmonary tuberculosis.

DIMINUTION IN SIZE. Small development of the bones is seen in idiots and cretins.

Rhachitis. In this affection the size of the body is lessened. For its distinction it is important to know how rapidly the osseous deposits in childhood have formed. The fontanelles and the epiphyses must be examined. If the fontanelles are open beyond their period of closure in health, or if the epiphyses are enlarged and lack firmness, the condition points either to simple malnutrition or to an affection of the bone known as rhachitis. In rhachitis late development of the teeth is observed. If at the same time the ribs are examined, nodules will be detected at the junction of the bone with the cartilage. These may be seen, as well as felt, if the child is thin. They form the so-called *rhachitic rosary.* The thorax also is changed in shape. At the junction of the cartilages and ribs a depression takes place which is continuous

with a groove which passes out from the ensiform cartilage toward the axilla. This transverse curve is known as Harrison's groove. It may deepen with inspiration. At the same time the sternum projects, forming the so-called "pigeon-breast" (see Thorax). On examination of the long bones changes are noticed at the lower end of the radius and ulna, and sometimes at the end of the humerus. The parts are enlarged at the junction of the shaft and epiphyses. There may be thickening of the clavicles at the sternal ends. In the legs the lower end of the tibia becomes enlarged, and at times the upper end, or even the shaft, becomes thickened. The child becomes bow-legged, or the tibiæ and femora may arch forward. Knock-knee sometimes occurs. The bones of the vertebral column and of the pelvis are also affected. The spine is usually curved posteriorly, but lateral curvature may also be produced with it. The contraction of the pelvis is such as to narrow its outlet—a matter of much importance for the future of female children.

The head of the child with rickets is quite characteristic. It has been mentioned that the fontanelles remain open for a long time, and areas of ossification are imperfect, so that the bone yields to the pressure of the finger. This occurs particularly at the side, and the term craniotabes is applied to it. The large head is square in shape when looking over it from above downwards. It gives to the face a peculiar appearance. It is proportionately very small, especially in the lower two-thirds, while the forehead is broad and square.

The condition is not difficult of recognition if the general and local appearances just indicated, associated with the symptoms of the disease (see Rhachitis), are coupled together.

Osteomalacia. Among the general affections of the skeleton, which may cause lessened size, *osteomalacia* must not be forgotten. As the lime salts are dissolved, the bones become preternaturally soft, break on the slightest provocation, or bend in various directions, depending upon the external pressure and the direction of the muscular force. The ribs are drawn in by inspiratory forces until the cavity of the thorax is lessened to a degree incompatible with life. The pelvis is deformed so that labor is impossible. (It occurs frequently in pregnancy.) All sorts of fixed contortions are assumed. If able to be up, the body shortens, the back becomes rounded, the neck stooping so that the chin is brought close to the sternum. On palpation, the bones can be indented by the finger, and crepitate like eggshells.

Osteomalacia is easily distinguished from carcinoma or sarcoma of the bones. In the latter spontaneous fracture occurs in various parts of the skeleton, but is generally preceded by pain and swelling at the seat of fracture. Then, in sarcoma subcutaneous hemorrhages are present. When one joint is affected, osteo-sarcoma, the same eggshell crackling is observed.

6. **The Exterior in General. The Skin.** The external examination reveals the color of the skin, its tone, the degree of moisture, the presence of eruptions, of hemorrhages, and of scars. The temperature is also observed.

THE HUE AND COLOR. The portions exposed to the air exhibit more varied and pronounced changes of color than parts that are cov-

ered. It is understood that the changes in color herein described refer more particularly to the face and hands, and that the color of other parts partakes of the same tint as that of the face, other things being equal, except that the intensity is less. Comparison of the two should always be made, and the mucous membranes examined, as control experiments. For the latter, the conjunctivæ, lips and mouth are sufficient, always remembering the possibility of hyperæmia of the conjunctiva from other causes.

Local change of the face will be particularized in this section. It is not to be forgotten that the color varies with the type, whether blonde or brunette, and that variations in the latter at times easily escape recognition.

The skin in a healthy child is of a faint pink color; as age advances it loses its fresh appearance and becomes paler, except in those whose occupation exposes them to atmospheric influences. In the latter the skin becomes weather-stained, and may assume a mahogany or reddish-brown hue. In old age the color is apt to deepen and become duller, while the loss of subcutaneous fat allows the skin to lay in folds, especially about the jaws and neck, and wrinkles are marked, especially between the eyebrows, over the nose, and at the angles of the eyes and mouth.

Apart from these changes, which are physiological or those necessarily the result of occupation, the skin exhibits changes the result of the habits or health of the individual. Some persons, especially if blondes, retain to old age the fresh, clean, pink skin of childhood. In others is seen early a dull, *muddy complexion.* This is common in those who use coffee to excess and are of constipated habit. In others, digestive derangements, particularly constipation, produce in addition to a muddy complexion, crops of acne and comedones or black-heads. It must be admitted, however, that some persons preserve a fresh complexion in spite of marked digestive disturbance. Considerable congestion of the superficial bloodvessels, giving the person a *florid* appearance, may be due, especially in a young person, to alcoholic excesses; and there is a popular belief which connects such an appearance, when coupled with a tuberous nose and a crop of angry-looking pustules, with a prolonged use of spirits.

Color Increased. The abnormally red skin. Physiological hyperæmia has been spoken of. The color is intensified when the capillaries are overfilled, or the blood current is unusually rapid. The hyperæmia may be general or local, and is due to dilatation of the capillaries, possibly from nerve influences. *General* hyperæmia is seen in fever and in poisoning from atropine. It is the glow that the warm bath and external friction excite. *Local* hyperæmia attends the phenomena of blushing and comes and goes in nervous persons, with every psychical impression. Abnormal redness may be diffused over the whole face or may present the circumscribed flush of phthisis; the local deep-red area, on one cheek, of pneumonia; the evanescent flush of anæmia, with cardiac palpitation; and the creeping flush, with raised border, of erysipelas, appearing on the bridge of the nose or at the nostril. In phthisis small excitement or exertion, taking food, or the onset of fever, tinges the cheek

with the blush of hectic. In migraine, the burning flush may be limited to one side. Capillary congestion on the cheeks or tips of the nose occur with the endarteritis of the aged, but is seen also in earlier life in cases of hepatic cirrhosis, or obstruction to the hepatic circulation from other causes.

Color Lessened. Pallor. It is caused by diminution in the amount of blood in the capillaries, or because the richness of the blood in hæmoglobin has been reduced.

Diminished amount of blood in the capillaries occurs from active contraction or spasm of the arterioles, from hemorrhage, or from weak heart. The pallor that arises, therefore, is usually *acute* or *temporary*, and may be recurrent. It results from fright, syncope, or nausea and vomiting. It occurs also in acute poisoning, in acute diseases, such as diphtheria, and in hemorrhage. The pallor that arises from hemorrhage comes on more gradually, that is, in the course of an hour or more, or during three or four days. Of course, if the hemorrhage is excessive the pallor may come on in a few minutes. Sudden pallor in the course of diseases which may be attended by hemorrhage, is of diagnostic significance, as in the course of aneurism, gastric or intestinal ulcer, and the ulceration of typhoid fever. With the onset of the pallor, if due to hemorrhage, the symptoms of collapse are seen.

Pallor of long duration, or *chronic pallor*, if we may so term it, is seen in a number of diseases. In all of them there is diminution in the amount of red corpuscles, and destruction of the hæmoglobin. It is characteristic of blood affections, as the forms of anæmia and leucocythæmia. It is seen in striking form in chronic Bright's disease, in cancer, in chronic poisoning, as from lead or arsenic, in chronic catarrh of the stomach or of the bowels, and in chronic infectious processes, as tuberculosis and syphilis.

While paleness is recognized as the fundamental or prevailing color of the skin in many of the above-noted affections, a further tinge gives a characteristic hue to the skin; thus in *chlorosis* there is a greenish appearance of the face, which is in striking contrast to the pearly colored conjunctivæ. In *carcinoma*, the yellowish tinge to the pallor often causes it to be mistaken for jaundice. In *pernicious anæmia*, a straw-colored appearance of the skin has been frequently described on account of which cases have been thought to be due to carcinoma. It is worthy of remark that the cachectic pallor in carcinoma is not likely to occur unless there are primary or secondary deposits in the gastro-intestinal tract or the liver, and it is well known that pernicious anæmia is usually secondary to gastric or hepatic disorder. The peculiar hue of the pallor, therefore, may have a common cause in these affections. The pallor that attends *Bright's disease* is usually associated with slight puffiness under the eyelids or local dropsical accumulations elsewhere. In chronic poisoning with *lead*, pallor is associated with a blue line upon the gums, and drop-wrist; while in arsenical poisoning there is frequently associated a puffiness of the eyelids and looseness of the bowels.

It is not well to lay much stress upon the variations in hue of the pallor. They are not of diagnostic importance in themselves, but only

when associated with the characteristic symptoms and signs of the respective affections in which this hue occurs.

It must not be forgotten that there are a large number of individuals in whom pallor is the normal condition. This is particularly the case with those who lead a sedentary life, and are confined within doors. There are a number of occupations which predispose to pallor.

Jaundice. Jaundice is a symptom due to a number of diseases. In the first place it is most frequently due to disease of the liver, and this variety is known as hepatogenous jaundice. It may also be due to destruction of the corpuscles of the blood and liberation of the hæmoglobin, and then is known as hæmatogenous jaundice. The various causes of the former will be considered under diseases of the liver. The latter form is due to destructive agencies in the blood, such as ptomaines, which are absorbed in gastro-intestinal disease, or poisons that develop in the course of pyæmia, yellow fever, malarial and relapsing fevers; it may also be due to snake-bite or to poisons that are imported, as in mineral poisonings.

In both instances, the yellow coloration of the skin is due to coloring matter of the bile in the blood, or bilirubin, which is deposited in the cells of the rete mucosum. The yellow coloration is seen not only in the skin, but in the conjunctivæ and other mucous membranes. The discoloration of the skin is not difficult of recognition. It varies in shades from a slight yellow hue to yellow-green, and in many forms of jaundice to brownish-yellow. The yellow hue of the skin in jaundice may be preceded by tinging of the conjunctivæ, and if the former is doubtful, it can be corroborated by the appearance of the mucous membrane. The mucous membrane under the tongue early gives evidence of jaundice. Or if the lips are everted and a glass slide pressed evenly on the surface, the yellow discoloration of the mucous membrane will shine through.

The yellow tint of the conjunctivæ must not be confounded with the same color due to sub-conjunctival fat. The latter is not uniform in the conjunctivæ, and may be seen to occupy cone-shaped areas.

The physiological yellow color of the skin that is seen in infants shortly after birth is not a true jaundice, but in all probability arises from excessive destruction of red corpuscles in the over-congested skin. On light pressure with the finger the color changes. It fades from shades of yellow into the genuine flesh-color. The conjunctivæ are natural, and the urine is free from bile pigment. The fæces are normal. By these symptoms a distinction can be made.

While jaundice is a symptom, it is nevertheless the cause of many symptoms, the presence of which may be of diagnostic value in determining the nature of the yellow color of the skin in cases of doubt. The *yellow coloration* of the conjunctivæ and the mucous membranes has been mentioned. (1) *Itching.* In addition, the surface of the body is often seen to be covered with scratch-marks, due to itching, caused by irritation of the peripheral ends of the nerves in the skin by bile pigment. (2) *Slow pulse.* Slowness of the pulse also frequently attends jaundice. The coloring matter invades the fluids of the body and is carried off by the kidneys. (3) *Secretions* and *excretions.* The

saliva, or expectoration if present, is bile-tinged and the *urine* is dark-colored, due to the presence of the pigment. (See Urine.) While the excretions are all tinged with bile in the hepatogenous form, the fæces are free from bile, hence they are pale or of an ashy color. On account of the absence of bile in the intestines its physiological purposes are lost, and therefore *flatulency* from fermentation becomes an important symptom.

Cyanosis. This peculiar hue is recognized without difficulty. The bluish or bluish-red appearance of the skin is first seen at points farthest from the central organ of circulation, as in the extremities. The mucous membranes, in which the capillary circulation is readily seen, also exhibit the change early. Hence the blueness of finger-tips, particularly underneath the nails, the bluish discoloration about the phalangeal joints, and the blue lips of the early stage of cyanosis. Thence the entire surface of the skin may become dusky or cyanosed as its cause increases in degree. It is not difficult of recognition. Its onset, it is said, can be anticipated by the state of the veins on the under part of the tongue; overfilling or extreme distention of these vessels always occurs in cyanosis. The color usually disappears on pressure at first wherever situated, but as the hue deepens it will remain in spite of pressure.

Causes. Cyanosis is due (1) to overfilling of the veins and capillaries with blood not sufficiently oxygenated, or (2) to an excess of venous blood, oxygenation not being interferred with.

1. All conditions which interfere with the aëration of the blood lead to the development of cyanosis. Obstruction of the air-passages, or encroachment upon the extent of respiratory capacity, or interference with the circulation in the lungs, will cause this condition.

a. Obstruction of the air-passages. This may occur in the upper respiratory tract, or in the capillary bronchi. Faucial obstruction, on account of abscess or tonsillitis, or in rare cases diphtheria, causes moderate cyanosis. Affections of the larynx which cause obstruction, produce cyanosis varying in degree with the amount of obstruction and its persistence. The cyanosis is of short duration in spasmodic croup, and in laryngismus stridulus; it is prolonged in the more persistent inflammatory affections. Its onset, in moderate degree, as seen by the purple lips or dusky finger-tips, is of serious prognostic import in the course of tuberculous laryngitis, even if symptoms of grave obstruction have not arisen. Tumors, pressing on the trachea or bronchi, narrowing the air channel, cause cyanosis. The tumors may be situated in the neck, as the thyroid gland, or within the mediastinum. Spasm of the bronchi, as in asthma, occlusion of the bronchioles, as in bronchitis, both acute and chronic, and particularly the grave form of capillary bronchitis seen in childhood, cause cyanosis.

It must not be forgotten that foreign bodies anywhere in the course of the respiratory tract in its upper regions are fruitful sources of cyanosis.

b. In encroachments upon the normal air-space, such as take place in pneumonia, in œdema of the lungs, in tuberculosis, in all forms of pleural effusion compressing the lung, and in conditions beyond the thorax which interfere with expansion, lead to the development of

cyanosis. Deficient expansion, and therefore lessened respiratory area, is of common occurrence in affections which interfere with the action of the respiratory muscles. This interference may be either on account of paralysis or on account of pain, or, in the case of the diaphragm, on account of pressure from fluids or accumulations in the abdominal cavity underneath. Large peritoneal effusions, and abdominal disease, causing enlargement with upward pressure of the diaphragm, produce it. In bulbar paralysis and peripheral neuritis, in paralysis of the diaphragm, in spasm of the muscles of respiration, as in tetanus, it is seen. In forms of progressive muscular atrophy cyanosis is also observed, and in other rare affections of the muscles, as trichinosis.

c. Interference with the circulation within the lungs, from pressure on the bloodvessels, pulmonary artery or vein, or from diseases of the heart itself, is a most frequent cause of cyanosis. In affections of the heart it is not seen until, in the case of valvular disease for instance, compensation is lost and the blood is accumulated in the lungs on account of dilatation of the right heart. It is seen that in the latter, both conditions are combined and contribute to the cause of the cyanosis; that is to say, in affections of the heart or obstruction of the pulmonary vessels, the congestion of the lungs that ensues is also associated with more or less obstruction of the bronchi on account of collateral congestion and catarrh.

2. *Obstruction to the flow of blood* anywhere in the circulation will lead to the development of cyanosis. This is the cyanosis of passive congestion. Cyanosis originating from causes mentioned above is always general. Cyanosis that develops from causes which will be indicated in this section may be general or local, depending upon the seat of obstruction. If the heart is diseased and there is interference with the flow of blood through the aortic side of the circulation, on account of obstruction or regurgitation at orifices, the venous side becomes overdistended with blood. This form of cyanosis is typically seen in congenital heart disease. It occurs in valvular insufficiency, in disease of the heart muscle, and in pericardial exudation. The development of cyanosis in valvular heart disease always implies failure of compensation and dilatation of the organ.

Local cyanosis is seen in all cases in which there is obstruction of the venous trunks from external pressure, or from diseases of the venous wall causing thrombosis. It may be limited to the head and upper extremities in obstruction of the descending cava by tumor or aneurism, or to the lower portion of the trunk and extremities by obstruction of the ascending cava from tumors within the abdomen and thorax pressing upon it. One extremity may be the seat of local venous stasis from pressure upon the vein or its occlusion by thrombosis: the arm in cases of cancer of the breast and axillary glands, the leg in cases of femoral phlebitis, represent typical forms of venous stasis. (See under Fingers, Raynaud's Disease.)

The Bronzed Skin. The most marked form of bronzing is seen in Addison's disease. The external surfaces are changed in hue, and delicate portions of the skin underneath the clothing are also bronzed. The discoloration is not removed by pressure. The areas are irregular

in shape. The skin is soft and pliable. The pigment which causes the discoloration is deposited in the rete Malpighii.

The pigmentation is never seen in the cornea or in the nails. The axilla, the flexures of joints, the median line, the areola about the nipple and other normal areas of pigment deposit are the seat of this deposition of pigment. In the mucous membranes the bronzed areas are limited to patches; they are sharply circumscribed brown areas seen in the mucous membrane of the lips and cheeks.

The discoloration of the skin in Addison's disease must not be confounded with a similar discoloration that occurs on account of *sunburn*. The discoloration under the latter circumstances is limited to parts that are exposed to the sun, is more uniform, and the mucous membranes are free. Moreover, the anæmia and debility of Addison's disease do not attend it.

In persons living in filth a general discoloration of the skin takes place, known as "*vagabond's disease*;" but because it is so general and the skin is rough and thickened, and other evidences of filth are seen, it can easily be recognized. In the later stages of *jaundice* the dark-green or black hue of the skin might be taken for the general bronzing of Addison's disease. The appearance of the conjunctiva is sufficient to indicate the cause of the bronzing. In certain cases of *tuberculous peritonitis*, even if the capsule is not involved, the peculiar brown discoloration which simulates Addison's disease is present.

Uterine Chloasma. The pigmentation that occurs in uterine disease or in pregnancy frequently resembles the bronzing of Addison's disease. It is usually confined to the forehead and cheeks and the normal pigmentary areas of the skin. The mucous membranes are not affected, although in pregnancy there may be the characteristic change of the vaginal mucous membrane. In both, the general conditions that attend disease of the supra-renal capsule are absent.

The bronzing of Addison's disease, the pigmentation of "vagabond's disease" and of pregnancy must not be confounded with the discoloration—yellowish-brown in hue—of *tinea versicolor*, a parasitic skin disease. The latter is recognized by its color and irregular dissemination. It especially occupies the chest and spreads to the abdomen. It rarely ascends above the neck. It does not usually, therefore, occur in parts exposed to the air, or in parts that are the seat of normal pigmentation. Then again, the surface desquamates in brownish scales. Examination of the scales put in a drop of dilute liquor potassæ under the microscope show both spores and mycelium. The spores are of the fungus microsporon furfur.

It must not be forgotten that there are cases of Addison's disease without the occurrence of the peculiar bronzing. The disease of the supra-renal capsule which is most frequently attended by the discoloration is tuberculosis. At times, the bronzing and other characteristic symptoms of the disease are associated with tuberculosis in other organs. Conversely, in cases of phthisis in which there is bronzing tuberculous disease of the supra-renal capsules may be suspected, and it adds to the gravity of the prognosis.

Argyria. If nitrate of silver is administered over a long period of

time fine black particles of the metal or of the albuminate are deposited in the kidneys, the intestine, and the skin. The corium is the principal seat of the deposition. The discoloration of the skin is gray or grayish-black. It is not changed by pressure, and is usually limited to the face and hands. Small specks may also be noted in the mucous membrane of the mouth. The cornea and nails are not affected. Persons are generally in good health, although the presence of the skin-change if seen in a patient with coma would point to the possible presence of epilepsy on account of which the drug had been taken.

Freckles. Freckles are not usually of special diagnostic significance. Their occurrence in an unusual degree has been observed, however, in cases of rheumatoid arthritis. Other signs and symptoms help to complete the picture of the disease.

The Nutrition of the Skin. The color, as previously indicated, is a fair index of the nutrition of the skin, but in addition to this palpation gives further information. In *health* the skin is smooth, firm, and elastic. When pinched between the thumb and fingers and then allowed to escape it slips quickly back into its former position. When pressed or squeezed it becomes pale from expression of blood, but resumes it natural hue immediately.

The readiness with which the blood returns after pressure gives information as to the character of the capillary circulation of the skin. This is active in health and sluggish in serious disease of the lungs, heart, and bloodvessels. In the eruptive fevers, especially in measles, scarlet fever and smallpox, sluggish capillary circulation with dusky eruption is a grave sign. In measles it is usually due to pulmonary complications, and in other infectious diseases to the overwhelming effects of the poisoning.

As age advances the skin becomes less elastic, and in old persons may lie in wrinkles. When pinched between the fingers the skin is more inclined to remain wrinkled. Fat persons whose skin is firm and hard are in much better condition than those whose skin is loose and flabby. The latter condition is frequently met with in babies, particularly those that are fed on artificial foods. When the skin is thin and dry and loses its tone, so that when pinched into folds it resumes its smoothness but slowly and sluggishly, it is usually evidence, in a person under fifty, of some grave cachexia, as carcinoma.

Moisture and Dryness of the Skin. Moisture and dryness are estimated with the tone of the skin, and in one sense are correlated. It is quite certain that when a skin is abnormally dry its nutrition is impaired.

In *health* the skin is not perceptibly moist, except as the result of physical exertion or under heat, or as the immediate result of imbibing a hot fluid or a sudorific drug. There is considerable individual difference, however, within the limits of the normal. Rheumatic and strumous persons may have a perceptibly moist and oily skin at all times, while others have a skin which perspires very little, even under influences which usually bring about perspiration.

Perspiration Increased. The term hyperidrosis is applied to this condition. It may be general or local. *A. General sweating* is seen

with normal or increased temperature. It occurs in the course of *rheumatism*, when the sweats are strong in odor and acid in reaction. It is seen in *tuberculosis*, especially the miliary variety. It is sometimes marked throughout cases of typhoid fever. General perspiration also attends the violent muscular action of *tetanus*, but is not seen in *epilepsy*. An example of general sweating is seen in that curious affection to which the term "sweating sickness" has been applied. It is a fever the nature of which is not well known, but in which this symptom is most pronounced. Sweating is extreme in *trichinosis*.

B. With subnormal or normal temperature. 1. *Sudden, temporary perspiration*. Sweats are seen in patients who are weak during the stage of *convalescence* from acute disease. In this period of disease sweats may occur suddenly, from a fright or shock, which under other circumstances would not influence them. General increase of perspiration may be of short duration and occur suddenly after fright or shock. It is the characteristic perspiration of *collapse*. The forehead is covered with sweat, large drops stand out on the face, the hands and feet are moist or wet with perspiration, and the whole surface of the body "leaks." In collapse that attends shock of all kind, or that occurs after hemorrhage or profuse discharge, as in cholera, this form of perspiration is seen. It is attended with a cold and clammy skin.

More striking still are the perspirations that suddenly break out in the course of acute disease, followed by a fall of temperature. We have (*a*) the critical sweats of pneumonia and relapsing fever; (*b*) sweat which terminates a paroxysm of intermittent fever; (*c*) the profuse perspiration that attends pyæmia, breaking out with each fall of temperature to disappear as it rises; (*d*) the night-sweats that attend tuberculosis and other exhausting diseases. In tuberculosis, or when there is pus-formation, the oscillation of temperature, with or without chills, followed by the sweating, is known as *hectic*. Sudden breaking out of perspiration, general, but more notably seen on the face, attends dyspnœa of pulmonary origin and the attacks of dyspnœa in the course of organic heart disease. These perspirations are at times the result of an effort at elimination, on the part of the skin, to relieve the kidneys or bowels, such as the perspiration of *urœmia*, which is attended by a urinous odor. At times in jaundice it may also occur.

In the conditions just mentioned there is coolness of the skin, and especially of the extremities.

2. *Prolonged Perspiration*. In exhausting diseases general persistent perspiration may occur, particularly in the later stages, as in tuberculosis, and in any disease attended by persistent dyspnœa.

Local increased perspiration (*hyperidrosis localis*) occurs when there is local vasomotor paresis. Thus, in organic diseases of the brain and in affections of the peripheral nerves, in some forms of neuralgia, it has been observed, and in migraine with hysteria. Sometimes one side of the body alone is affected, even in a malarial paroxysm (hemidrosis). Unilateral sweating of the head arises from pressure on the sympathetic nerves in thoracic aneurism.

Local sweats are sometimes significant. This is the case particularly with a sweat confined to the head, which occurs usually in children, and

is one of the striking characteristics of rickets. With the local sweating the patient rolls his head at night on account of the discomfort. The hair on the back of the head is seen to be rubbed off.

Diminished Perspiration—Anidrosis. The skin is abnormally dry in the early stages of acute disease attended with fever, particularly if the febrile rise takes place suddenly, as in the acute digestive disorders of children. In adults when the disease is accompanied by high fever, as in thermic fever, the skin is dry. In the first day of the eruption of the exanthemata the dryness is marked. Dryness of the skin is of frequent occurrence when there are copious discharges of water from the bowels or the kidneys. In choleraic diarrhœa the dryness occurs suddenly. In some affections, as diabetes and Bright's disease, the dryness extends over a long period of time, and is frequently attended by eruptions or desquamations and by the formation of boils. When there are accumulations of serum in the lymph spaces of the subcutaneous connective tissue, or changes in the connective tissue, as in dystrophies or myxœdema, the skin is dry because of the stretching and pressure on the bloodvessels.

Scars. Scars are important proofs of the occurrence of previous disease, especially smallpox, chickenpox, and syphilis. Scars of the first two occur in the form of circular pits, and almost always on the face. Scars of syphilis are larger, circular or oval in shape, and seen usually to best advantage on the extremities, but the single scar on the forehead is strikingly suggestive. Scars upon the legs in persons under thirty years of age, when not traumatic, are almost always syphilitic. Scars as the result of suppurating glands are seen most frequently in the neck, but may be found wherever there are glands, especially under the jaw and in the axilla and groin. They are most liable to occur in tuberculous persons, either spontaneously or as the result of the exanthemata, erysipelas, or other infectious disease. When such scars are met with in a person with incipient tuberculosis the prognosis becomes more anxious.

The appearance of the scar indicates in a general way its age, and hence throws light upon the patient's previous history and also serves as a check upon the accuracy of his statements.

Scars the result of wounds, injuries or operations may be seen anywhere; they are of importance only so far as they may furnish a clue to the cause of existing disease. Of such nature are the scars upon the head in cases of brain disease, particularly epilepsy.

The scars of pregnancy, the striæ seen upon the lower part of the abdomen and the upper part of the thigh, must not be confounded with similar scars that occur in great œdema, and which are sometimes found in fat persons.

Hemorrhages. Hemorrhages in the skin are called, according to their size, *petechiæ*, *ecchymoses*, *vibices*, and *hæmatomata*. The petechiæ and ecchymoses are apt to appear in the hair follicles, and vary in size from a pin-point to a split pea. They must be distinguished from erythematous and other eruptions.

Mode of Recognition. They may be raised above the surface of the skin; they do not disappear upon pressure, and vary in hue from deep red to yellow-brown, according to their depth beneath the surface and

to the degree of absorption that has taken place since the hemorrhage occurred.

Vierordt advises the following test to distinguish them from erythemas: Press a piece of glass (a microscope slide) upon the suspected spot. A hemorrhage is rendered more distinct, while the surrounding part becomes more anæmic. An inflammatory hyperæmia, on the other hand, disappears.

Cause. They may be due to affections of the blood or disease of the bloodvessels. When they occur in the course of blood diseases it is because there has been such a change in the quality of the blood that diapedesis can take place more readily. They are more particularly, but no exclusively, seen in dependent parts, especially the lower extremities.

Significance. While the recognition of subcutaneous hemorrhages is comparatively easy, their diagnostic significance must depend upon the phenomena with which they are associated, or upon their occurrence conjointly with hemorrhages from other organs. Moreover, the *situation* of the hemorrhage is in a measure an index as to its causal origin; thus hemorrhages about joints are purpuric or hæmophilic.

1. *Hemorrhage with Fever* If subcutaneous hemorrhages are found in the course of acute disease with high temperature they may be dependent upon changes in the quality of the blood, or upon obstruction of the bloodvessels with emboli. The former class are seen in cerebro-spinal fever, and in measles, variola and scarlatina. The cerebral and spinal symptoms in the first affection point to its probable origin. In the exanthemata they develop with the characteristic eruption, although the latter may be darker in color than normal. Hemorrhages will probably take place at the same time from the mucous membrane, hence the nares will be occluded and the mouth and fauces filled with clotted blood. In milder degree sordes collect in the mouth. They usually indicate malignancy in these affections.

The latter class of hemorrhages are hemorrhagic infarcts and are seen in pyæmia and ulcerative endocarditis. The hemorrhages are small, sometimes elevated, more abundant on the extremities, but distributed over the trunk; they are seen as small areas in the mucous membranes, observed in the conjunctivæ, and on ophthalmoscopic examination found in the retina. The association of chill, fever, and sweat, the presence of pus in some structures of the body, and the characteristic joint affections, point to pyæmia. On the other hand, if due to ulcerative endocarditis, the physical signs of this affection render the recognition of the cause of the hemorrhage clear. Finally, in fever with involvement of the joints, of rheumatic in contradistinction to pyæmic origin, we have the occurrence of purpura. In the most marked degree it is seen as peliosis rheumatica, and is associated with hemorrhages in other portions of the body.

2. *Hemorrhage with Anæmia.* In all forms of anæmia attended by debility hemorrhages occur. In idiopathic or pernicious anæmia they are usually only small hemorrhages, but may become more extensive. They occur on the extremities, and usually on the dorsum of the feet or hands. There may also be retinal hemorrhages. In the secondary anæmias that arise in the later stages of carcinoma with emaciation, particularly of the stomach, in the later stages of Bright's disease, and of cirrhosis of the liver, they are also seen.

Purpura Rheumatica. If the hemorrhages are limited to the legs, and particularly if found about the joints, and if they are comparatively large, having the appearance of black-and-blue spots ranging from the size of a three-cent piece to a half-dollar, if there has been a history of rheumatism, or the patient complains of joint symptoms, they are usually of the nature either of rheumatic purpura or purpura hæmorrhagica. Some forms of purpura, as peliosis rheumatica, are attributed to the presence of bacteria, and indeed, with scurvy included, are by some writers said to be infectious. The micro-organism, however, has not been isolated. Finally, reference must again be made to the subcutaneous hemorrhages which occur in sarcoma of the skin and bones, and in *jaundice.* In the latter affection when malignant, the mucous membranes also bleed. The lips, gums, and tongue are covered with sordes. The conjunctiva is the seat of hemorrhage, and so also are other mucous membranes.

Scurvy is an affection characterized by anæmia, debility, and wasting, in which there are hemorrhages under the skin as well as from the mucous surfaces. The gums are particularly affected. They bleed easily. Hemorrhages also occur in the deep lymphatic spaces, in the muscles, underneath the periosteum, and in the joints.

3. *Subcutaneous Hemorrhage with Hemorrhage Elsewhere.* The diagnostic significance of hemorrhage under the skin is clearer when associated with profuse hemorrhages in other portions of the body, and when also there is a history of the occurrence of such hemorrhages in the family. The peculiar disease, *hæmophilia*, is attended by hemorrhages without cause, and with the peculiarity that for successive generations bleeders belonging to the male sex have been found, the disease being transmitted through the female members of the family.

4. *Hemorrhage in Central Nervous Disease and Neuritis.* Mitchell has written of the neurotic origin of purpura. Subcutaneous hemorrhages are seen in neuritis.

Eruptions. Diseases of the skin are usually characterized by eruptions. Now, such eruptions may be primary and local (from causes operating directly on the skin) in the sense that they occur independently of any internal affection; or secondary, the resultant of an internal morbid process. The morbid process in each does not differ, nor do we have morbid processes in the skin that differ, from the same in other epithelial structures. The anatomical and physiological peculiarity of the part causes the difference in the phenomena. Hence anæmias and hyperæmias, inflammations, acute or chronic, with or without exudation; hemorrhages, atrophies, and hypertrophies, new growths and parasitic affections are found. But instead of a painless inflammation with transudation of mucus, as in mucous membrane inflammation, we have a more or less painful inflammation, with itching (nerve supply) and with sebaceous and sudoriferous gland exudation. Otherwise the same symptoms attend each, but ocular examination of the bronchial mucous membrane is not possible.

Mode of Recognition. We recognize the process in the skin by *inspection*, and differentiate the processes by inspection and palpation.

While reference must be made to special works on skin diseases for a description of the primary or local skin affections, the secondary affec-

tions will be briefly noted. It must not be forgotten that the local affections—eczemas, parasitic disease, etc.—are modified by the general condition or state of health of the patient.

Clinical Significance. This depends, first, upon the special character of the eruption, the nature of the lesion; second, its distribution—(*a*) in the layers of the skin, (*b*) over the surface of the body; third, its association with other morbid phenomena or various circumstances.

I. *The nature of the lesion.* Observation concerning the nature of the lesion includes (1) its anatomical character, (2) the order of appearance, (3) its uniformity, and (4) the mode of invasion.

A knowledge of the anatomical lesions is essential in order to be able to define exactly the morbid process and apply the relationship of the lesion to the primary cause. For a long period of time the lesions have been divided into primary or secondary. The lesions known as scab, scales, raw surfaces, scratch-marks, and ulcers, are always secondary. Scars and maculæ appear latest. The other lesions herein described are primary. The writer follows Dr. Pye-Smith in the description of them, as well as in most of the matter appertaining to cutaneous affections.

1. *Hyperæmia, or congestion.*

a. Mere overfulness of the vessels from paralysis of the vasomotor nerves, with redness and heat, but without the exudation and tissue changes which accompany inflammation. This hyperæmic blush, readily produced in the physiological laboratory, is rarely seen as an uncomplicated morbid condition (*e. g.*, Trousseau's *tache cérébrale*).

b. Active, arterial, or inflammatory hyperæmia, varying in color from brilliant scarlet to rose-pink, and combined with heat, tingling, or other sensations.

c. Passive, venous, or congestive hyperæmia, dependent upon retarded circulation and distended venules. The color is purple, bluish, or livid, the surface is cold, and there are no painful sensations.

2. *Pimple, or papule.* A small, solid elevation of the skin.

a. The acute inflammatory papule.

b. The chronic large inflammatory papule, discrete or confluent.

c. A solid non-inflammatory papule.

d. Solid elevations of the skin, which may be called false papules.

3. *Vesicle.* A visible cavity in the skin filled with transparent liquid.

4. *Pustule.* A cutaneous abscess.

5. *Bulla, or bleb.* A very large vesicle.

6. *Scab, or crust.* A dried-up concretion of the contents of a vesicle, pustule, or bleb.

7. *Scale* (*squama*). A dry flake of epidermic cells.

8. *Wheal* (*pomphos*). A flat, solid elevation of the skin, much larger than a papule, and of ephemeral duration.

9. *Scratch-mark.* An injury to the skin, of linear form and curved outline.

10. *Raw.* A surface which has lost its horny layer of epidermis.

11. *Chap* (*rima*). A crack or fissure which goes through the epidermis.

12. *Sore* (*ulcus*). The result of destruction by inflammation, which has reached below the Malpighian layer and has destroyed the papillæ.

13. *Scar* (*cicatrix*). The result of the healing process after an injury or disease deep enough to destroy the papillæ of the part.

14. *Nodule.* A solid elevation of the skin larger than a papule, and seated in its deep layer.

15. *Stain* (*macula*). A patch of increased pigmentation of the skin.

16. *Hemorrhage* (*ecchymosis*). When a bloodvessel of the cutis vera gives way, a dark red or purple mark is produced, which (like the macula) does not disappear on pressure.

The recognition of the exact anatomical lesion is not of sufficient purpose for diagnosis, unless at the same time the *mode of invasion* is observed. Often commencing at a focus, the rash spreads, or numerous foci appear and coalesce. The lesion is best studied in the more recently spreading part. Not only is the mode of local invasion to be noted, but also the *uniformity* of the anatomical lesion. Often, instead of a simple lesion, various kinds are present at the same time, or they develop in successive order, as in smallpox we have first the papule, then the vesicle, and finally the pustule.

II. *Distribution.* The location of the lesion in the various layers of the skin, and the distribution over the surface of the body, must be observed. The horny layers of the epidermis manifest pathological changes due to hypertrophy, atrophy, dryness or desquamation of the cuticle. Dead scales are the resultant, together with the hypertrophies and atrophies of the outline to follow (p. 92). The eruption in a large number of cases is limited to the living Malpighian layer of the epidermis and the papillary layer of the cutis. The hyperæmias (erythemata), and inflammations of all kinds, are confined to these layers. They never leave scars in this situation. The deep layer of the cutis is so intimately connected with the subcutaneous tissue that morbid changes in it involve the latter, and even extend deeper. The affections are more severe, but less numerous than affections of the superficial layers, and are always followed by cicatrices. The changes in the sweat glands, sebaceous glands, hair and nails, in so far as they refer to internal medicine, have been treated of in another section.

The occurrence of the eruption in different areas over the surface of the body is of great diagnostic importance in the various erythemata due to the exanthems, or to morbid conditions of the gastro-intestinal tract. The distribution will be noted in more detail when their eruptions are considered. The student should also bear in mind the relationship of eruptions or cutaneous changes of nutrition (trophic disorders) to the affected nerve supplies.

III. *Associate morbid phenomena.* The student of internal medicine should particularly observe the associated morbid phenomena, or concomitant circumstances, in order to determine the nature of the skin affection which is the expression of an internal disorder. The associated morbid phenomena of diagnostic significance are *fever, jaundice, albuminuria,* past or present *syphilitic* disease, *tuberculosis, rheumatism,* or the phenomena of the rheumatic habit. The presence of either one of these processes points to particular affections. Thus a large number of these eruptions are attended with fever; another group are of frequent occurrence in the course of rheumatism; another class

belongs to syphilis, while a fourth class is associated with anæmia, jaundice, or albuminuria. This subdivision is not on the basis of the nature of the eruption, but of its association with other phenomena. It will be learned later that all the groups belong to the hemorrhages or erythemata. The true relationship of the two classes of phenomena can be ascertained fully only by inquiry into the history and course of the eruption and of the concomitant phenomena. Thus, if the eruption is thought to be due to the exanthemata the period of incubation, mode of infection, symptoms of the invasion, and the progress of the attack must be inquired into.

General Symptoms. In order to determine accurately the cause of an eruption and appreciate its diagnostic significance, the general health must be inquired into, the condition of the stomach and bowels, and the character of the urine ascertained. It must be remembered that local skin disorders are influenced for good or ill by the general health. Functional disorders of the stomach and bowels are a frequent source of many of the erythemas, while in diabetes, pruritus and forms of dermatitis are of common occurrence. In Bright's disease also the latter are observed. The common cause for the eruption is the same in both, in all probability—that is, a perverted secretion of the skin, or, if œdema is present, impaired nutrition of the surface.

The *subjective symptoms* are of further importance in the effort to ascertain the true nature of an eruption. Pain, itching, burning, smarting, and tenderness are significant of the inflammations. But, in addition, *pains* different from those which attend inflammation are present and characteristic. They are of a neuralgic nature, and while intermitting they are not limited to the area of the skin affection. They are distributed in the line of the nerve trunks of the adjacent regions. They often precede the development of the eruption. Pain of this character is seen in herpes zoster. *Itching* is an important symptom in disease of the skin. It is not present in the eruption due to the exanthemata generally, except in smallpox and rubella. Its absence is a striking peculiarity of the eruptions of syphilis; but in erythema, especially if associated with œdema, it is a most annoying symptom. Its presence in other skin diseases, as eczema, psoriasis, and the parasitic affections, is so much more common and of such extreme degree of annoyance that we may be safe, in the determination of the nature of an obscure eruption, in excluding the class which is particularly associated with internal diseases, by the presence of this symptom.

Itching may be present without anatomical evidence of skin disease. It is seen in the troublesome *pruritus* that occurs in the aged, particularly about the intestinal and genito-urinary orifices, symptomatic of affections of the organs related thereto. It is a symptom which should lead to an examination of the urine, as diabetes is sometimes found to be the fundamental source of the complaint. It has been previously noted that in jaundice, itching to a high degree occurs. It is also due to the internal administration of drugs, as opium and morphine, and sometimes quinine.

In addition to the associate pathological phenomena which should be ascertained in the study of skin eruptions, in order to determine their

relationship to internal affections, other circumstances should be inquired into, such as the occupation, the character of the clothing, degree of cleanliness of the patient, the effects of climate, including seasons, temperature, and state of the air.

In order more thoroughly and yet in a concise manner to appreciate the various skin eruptions and their pathological relationship, the following outline is included from the concise work of the author previously mentioned, to whom the writer is indebted for much of the data of this section. A study of the table likewise shows at once the relationship of the eruption to the internal disorders which concern us more particularly in this work:

DISEASES OF THE SKIN REGARDED AS PHYSIOLOGICAL PROCESSES.

(*Pathological Arrangement.*)

Acute Inflammations.—Diffuse, *e. g.*, scarlatina, morbilli, syphilis, roseola (eruptive fevers; erythema).
With venous congestion—Erythema nodosum (rheumatism).
With œdema—Urticaria, erythema nodosum (gastro-intestinal disorder and rheumatism).
With necrosis—Furunculus, anthrax (diabetes).
Localized in papules—Enterica (erythemata), syphilis, eczema, prurigo.
Localized in vesicles—Eczema, zona, variola, scabies, herpes, varicella (eruptive fevers, infectious diseases).
Localized in pustules—Impetigo, variola, scabies, syphilis, sycosis, acne.
Localized in blebs—Pemphigus, scabies, rupia.
Desquamating during involution—Scarlatina, etc.
Chronic Inflammations.—With venous congestion—Acne rosacea, pernio.
With over-production of epidermis—Psoriasis, pityriasis rubra.
With œdema—Elephantiasis.
With fatty degeneration—Xanthelasma.
With hypertrophy—Elephantiasis.
With cicatrization—Cheloid.
With ulceration—Lupus, syphilis, lepra.
New growths—Xanthelasma, lupus, lepra, syphilis, cancer.
Atrophy—The senile skin, lineæ gravidarum.
Hypertrophy—Ichthyosis, cornu cutaneum, clavis, verruca.
Hemorrhage—Traumatic (*e. g.*, flea-bites), typhus, scurvy.
Pigmentation—Syphilitic maculæ, melasma, chloasma, icterus, ephelis.
Congenital malformations—Ichthyosis, cutaneous nævus.
Neurosis—Pruritus (diabetes, jaundice).
Anomalies of Secretion.—Increased, diminished, or perverted—Seborrhœa, xeroderma, hyperidrosis, anidrosis, chromidrosis, etc. Obstructed—Comedo, milium, acne; sudamina.

A glance at the above outline will show that the eruptions which particularly concern us belong to the class of diseases to which the term *erythema* is applied.

ERYTHEMA. 1. *Classification.* Erythemata may be divided, in accordance with the classification of Kaposi, into acute, contagious, exudative dermatoses, represented by measles, scarlatina, rubella, and smallpox; and the acute, non-contagious, inflammatory dermatoses, which may be further subdivided into: First, typical forms, idiopathic and toxic, including urticaria, or nettle-rash; second, varieties of herpes; third, erythema due to boils, colds, or erysipelas. The first group of the *non*-contagious form includes the class which should always be considered in

connection with the diagnosis of fevers. The skin inflammations closely simulate the eruptive fevers as to the eruption, the fever and even the affections of the mucous membranes. Besnier has named them the pseudo-exanthems, and divides them into the rubeoloids and scarlatinoids. Both simulate eruptive fevers throughout their course, and hence both are acute and febrile. The scarlatiniform erythemas are febrile at the beginning, subacute in course, but of longer duration than fevers they simulate. They are the most common forms, and arise from infectious diseases, such as puerperal fever, septicæmia and gonorrhœa, or from toxæmia due to drugs or articles of food.

2. *Character of eruption.* The erythemata are characterized by (*a*) rose rash with injection of the surface, either (*b*) with general œdema, or with circumscribed local œdema, forming wheals, or with papules. In rare forms bullæ are also formed. (*c*) The rash is followed by a branny desquamation. (*d*) The exudation that attends the lesion is always watery, in contradistinction to the sero-purulent or purulent exudation of eczema and scabies. Sometimes slight hemorrhages attend the lesion, as in cases of purpura or of urticaria. (*e*) The *course* of the erythema is of diagnostic significance. It begins quickly and is usually attended with febrile symptoms, sometimes mild, again very intense. (*f*) The duration is short; at least it is not indefinite. The erythemas that are recurrent must not be considered to be one process of long duration. (*g*) The locality of the erythema is not of precise diagnostic significance. The eruption is usually symmetrical, and the favorable localities may be defined as the extensor surfaces of the forearms and leg, the face, cheeks, and neck; and, thirdly, on the chest and abdomen. True erythema does not attack the scalp, the flexures of the joints, the palms (except erythema multiforme) and the soles. (*h*) The local symptoms that attend erythemata are mild. Local tenderness is more marked than in eczema. Smarting and tingling are complained of, but severe pain and excessive itching are rare. Only when wheals are present do we find pruritus. The rash of erythema does not spread. Patches occasionally unite, but an affected area never enlarges its borders.

3. The *ætiology* of erythema is involved in obscurity. Although the frequent associate phenomena are not of ætiological, they are certainly of diagnostic significance. We may have them occur under the following circumstances: 1. In one class the eruption is symptomatic, depending upon dyspepsia or upon rheumatic fever. 2. In the eruptive fevers, especially scarlatina and measles, in enteric fever and cholera, and in syphilis, there is an early erythema preceding the later true eruption. 3. The most striking instance of the relationship to internal disorder is seen in the rash that arises after the administration of medicine, as copaiba, or after the taking of certain foods. 4. The erythemata occur most commonly in children and young people. They are very frequent in men. The age at which they occur coincides with that of rheumatism.

4. *Varieties.* The following are the varieties of erythemata: First, *erythema multiforme* in simple form, with papules or with exudation; it may disappear in a few hours, or persist for a day or two and form rings (*erythema fugax* and *erythema annulatum*). With the fading of

the redness faint desquamation follows, and there may be a few pigment marks. The annular form is observed in rheumatic fever. In addition to rheumatism as the cause of erythema multiforme, it may be found associated with the following affections: Typhoid fever, puerperal fever, gonorrhœa, cholera, infectious endocarditis and osteomyelitis, syphilis, leprosy, vaccination, and surgical septicæmia.

Erythema lœve often appears upon the tense skin of dropsical parts. It may be the result of acupuncture.

Vesicular and bullous erythema. To this class belong the affections known as herpes and erythema bullosum. *Herpes zoster* is observed in the cutaneous distribution of one or more nerves. It consists of vesicles of flattened form ranged in clusters of twenty or thirty lying on a reddened, slightly swollen bed of skin. The number of clusters varies from one to ten. The vesicles develop in quick succession, beginning usually at first nearest the roots of the nerve whose branches they follow. A short papular stage precedes the vesicles, and some of the vesicles abort. The eruption tends to dry up in five or six days. The crusts form in yellowish or brownish clusters, which fall off in the third week, leaving purple stains.

When the disease attacks the face the fifth nerve is followed in its course. The several twigs of the trifacial are traced out from their points of emergence from the bony canals. Great swelling of the eyelids sometimes takes place on account of the loose tissue, so that the lesion may be mistaken for erysipelas. Ulceration of the cornea and iris sometimes occurs, and when lower divisions of the trifacial are affected, vesicles may appear in the mucous membrane of the mouth and palate. The cervical nerves and those of the upper extremity are also affected in their distribution. The eruption on the arm rarely goes below the elbow. When the second and third intercostal nerves are affected, the intercosto-humeral branch produces an eruption down the inner side of the arm. The eruption occurs frequently on the trunk. Following the course of the dorsal nerves it slants downward as it approaches the pubes.

In the distribution of the disease in the lower limbs the eruption rarely extends below the knee or buttocks. It follows the course of the external cutaneous or anterior crural nerves, or that of the small sciatic. Some of the branches of the sacral nerves are also affected. The disease is unilateral, and its precise limitation to one-half of the body is of the greatest diagnostic significance.

While fever or general symptoms do not usually attend its course in any extensive degree, insomnia and depression are likely to occur, probably on account of the severe neuralgic pain. Pain is the most important subjective symptom. It is localized in the nerves in the distribution of which the eruption takes place. It is not so likely to be present in the young. The pain may precede the eruption by several days, and persist long after the eruption subsides. This is particularly the case in old people.

Herpes labialis, or *facialis*, consists of vesicles arranged in groups or clusters upon a red patch of skin. They appear very suddenly upon the upper lip or the alæ of the nose; sometimes on the cheek or chin, and they may appear inside the mouth. They undergo some changes, as in herpes zoster, but are not attended by the neuralgic pain. They are

always symptomatic of an internal disorder, an acute catarrh (cold), or follow a rigor, as in intermittent fever or pneumonia. Herpes iris and herpes præputialis have no diagnostic significance of internal disease.

Erythema nodosum. With the erythema there is great œdema. The spots are somewhat painful and tender, but do not itch. The redness of the erythema is modified by the hue of venous congestion. Small hemorrhages may be seen. The patches develop on the legs, their long diameter being parallel to the tibia. They rise slowly into hard masses. They may be seen on the ankles or the calf, and sometimes on the ulna. They occur frequently in those who have suffered from rheumatic fever.

Urticaria is a form of erythema in which wheals, sometimes surrounded by an erythematous blush, are seen. It is an acute inflammatory œdema of the cutis. The serous exudation fills the lymph spaces and expels blood from the venules. It takes place suddenly, and may be excited by chemical irritation or a mechanical irritant, as the finger drawn across the skin. Small patches, or large white areas, are seen, due to the coalescence of smaller ones (giant urticaria). All parts of the body may be affected, except the scalp, face, and soles of the feet. The eruption is not symmetrical. Its course may be acute, or it may be chronic and transitory, characterized by successive attacks. It is the form of erythema in which intense itching is the most pronounced symptom. There are no other subjective symptoms. The itching causes restlessness and loss of sleep. Urticaria is symptomatic of gastric or intestinal disturbance, or the ingestion of drugs or poisons. Another form follows the tapping of a hydatid cyst. It occurs sometimes in women at each menstrual period, and may be traced to ovarian disorder. It may occur after severe shock to the nervous system, with high fever. It is not an infrequent complication of rheumatic fever. It occurs in men and women equally, but is most frequent in children and adolescents.

Medicinal Rashes. To the erythemata belong most of the so-called medicinal rashes.

The following drugs are known to cause erythema: bromide and iodide of potassium, copaiba, cubebs, the essential oils, capsicum, santonin, chloral, opium, morphine, antipyrin, salicylic acid and its compounds, iodoform, belladonna and atropine, tar, carbolic acid, arsenic, cannabis indica, digitalis, mercury, silver, and copper.

Belladonna produces in susceptible persons, or when administered in poisonous doses, a diffuse bright-red erythema, closely resembling that of scarlet fever, but without the darker red points which interrupt the latter. *Atropine* also produce in some persons, especially on the shoulders, arms, chest, and face, an eruption of disseminated, small, hard vesico-papules, showing no tendency to pustulation. They are seated on an inflammatory base, but are more superficial than acne.

The *bromides* produce a characteristic pustular eruption which is most intense upon the shoulders, face, chest, and arms. Large doses or long-continued administration is generally required to bring it out. It is conspicuous upon the face of some epileptics.

The *iodides* produce an eruption which is not frequently pustular,

but an erythematous rash is not uncommon. It appears chiefly about the forearms, face, and neck. Vesicles, bullæ, and purpuric spots are also occasionally seen.

The eruption produced by *quinine* is generally erythematous, and is attended with itching and burning; the face and neck are attacked first.

Opium and its alkaloid also produce in susceptible persons an erythematous scarlatinoid eruption which is accompanied with intense itching. Itching, especially about the nose, is much more common without an eruption.

Copaiba produces a vesico-papular or papular eruption which resembles urticaria and erythema multiforme. It is itchy. It is more apt to be seen on the extremities. It may be purpuric.

The eruption of *cubebs* is a diffused erythema, with millet-sized papules, coalescent here and there. Unlike the eruption of copaiba, it is more copious over the face and trunk than over the extremities.

Antipyrin causes a measles-like or urticaria-like eruption.

Erythemata of Infectious Diseases. The inflammations of the skin which are symptomatic of a specific infection are also of an erythematous variety. The term *exanthemata* has been applied to the latter, but the eruption of typhus and typhoid (enterica) belong to the same class. The characteristics and distinctions of the various forms will be described in sections devoted to the respective diseases. The student should remember the association with the general phenomena, particularly fever, the onset and course of which should be carefully observed. But to add to the confusion an erythema called roseola often precedes the fever.

Roseola. The rashes which precede the eruptive fevers are very liable to lead one astray as to the recognition of the true disease. Their association with this class of fevers has been indicated before. The form of erythema known as roseola, or rose rash, is especially seen. It is of a deep rose color, not arranged in crescentic patches, as in measles, nor scarlet and capable of being resolved into innumerable red points, as in scarlatina. It is not so diffuse as the latter. It precedes smallpox, scarlatina, measles, cholera, typhoid fever, syphilis, diphtheria, and malaria. In smallpox, in cases of cholera, and after parturition and surgical operations, the rash is copious, but is characterized by its being seated over the lower half of the abdomen and the anterior and inner aspect of the thighs. It may appear elsewhere, but is generally confined to that portion of the body. Erythema roseola may be mistaken for rubella, measles, or scarlatina. The following are points of distinction. First, it is not contagious and is not epidemic; second, there are no prodromal symptoms; third, the rash does not come out after a definite period of fever; fourth, it is not confined to any special locality of the body; fifth, the fever is of short duration and moderate degree, rarely above 101°; sixth, there is no catarrhal discharge from the eyes or nose or in the pharynx; the fauces and palate are reddened, without swelling; seventh, it is not seen in the mouth, like the eruptions of measles or scarlatina; eighth, the fever which precedes the eruption, if present, is of only a few hours' duration (in scarlatina it is twenty-four

hours, in measles seventy-two hours); ninth, the rash is not crescentic as in measles, or punctiform as in scarlatina, though it is to be admitted that severe cases of the affection cannot be easily diagnosticated, the development of the sequelæ alone concluding the diagnosis.

Sufficient reference has been made to the erythemata that attend *rheumatism*. A few other internal (infectious) disorders are associated with the development of an eruption. In *cholera*, during the period of reaction, a rose rash which may resemble erythema, urticaria or scarlatina appears coincidently with a rise of temperature. It is most frequently seen on the forearms and back of the hands, but may cover the back and limbs. It may be slightly hemorrhagic and last two or three days. A slight desquamation usually follows. In *influenza* a roseolous eruption, covering the trunk and limbs and becoming papular, is seen rarely.

In addition, in the course of *Bright's disease* erythematous eruptions are sometimes seen. Quite distinct from erythema læve, previously mentioned, two forms are observed, the roseola on the feet, legs and hands, rarely on the chest and abdomen, and the papular form on the thighs, arms and shoulders; itching and other subjective symptoms do not attend the eruption. A form with desquamation may begin on the limbs. These erythemata are common in the later stages of Bright's disease, but are not of ill omen. In acute Bright's disease a transient roseola is observed very rarely; so also is purpura. If there is much anasarca in tubal nephritis, erythema is more common. The eruptions generally appear independently of uræmic symptoms, and disappear during their continuance. They are allied in all probability with the inflammation which attacks the lungs and serous membranes in Bright's disease.

Sudamina. Here may be placed another eruption or condition of skin, common in the course of internal diseases. *Sudamina*, or *miliaria*, are small, clear vesicles seen in large numbers, generally on the abdomen, but also on any other part which reflects the light strongly. They are seen after the subsidence of anidrosis, when profuse sweats occur. While actual perspiration is seen on the forehead, the trunk may appear free from moisture. When the hand is placed over it, as on the abdomen, the dryness is noted, but at the same time a roughened, nutmeg-grater-like sensation is present. On close inspection this is observed to be due to the eruption just mentioned. The vesicles are usually of good prognostic omen in the course of febrile diseases, particularly typhoid fever. They are due to the accumulation of perspiration under the epidermis.

General Diagnosis of Skin Affections.

(*Condensed from* Pye-Smith.)

I. **Factitious Eruptions.** We must never forget the possibility of the affection before us being artificial. All kinds of dermatites, eczema, erysipelas, pemphigus, impetigo, may be simulated by the application of various irritants. Pigmentation also has been often imitated with success. Such artificial lesions will generally be found upon the arms,

rarely on the face, and scarcely ever beyond reach of the patient's hands. Mustard, cantharides, and some other irritants can be distinguished by help of the microscope.

II. Traumatic Eruptions. In all cases of dermatitis we should seek for the irritant, and sometimes it is so directly the cause of the disease that the eczema or impetigo in question may be considered purely traumatic, and efficient treatment immediately follows accurate diagnosis: *sublata causa tollitur effectus.*

Pediculi in the hair should be carefully looked for in all cases of impetigo in children, pediculi vestimentorum in prurigo of old people. The acarus of scabies, fleas, bugs, and gnats, should be looked for. In adults pediculi pubis may sometimes be found in the axillæ as well as in their proper region, and when they have been destroyed by mercurial ointment the patient is at once relieved from pruritus.

In many trades an irritant must be sought in the objects which the patient habitually handles. The coarser kinds of brown sugar are a frequent cause of eczema of the hands (grocer's itch). So with many of the "chemicals" used in a variety of modern handicrafts. Constant washing of the hands in washerwomen, in scrubbers, in potmen, and many others, produces eczema rimosum. The heat of the sun is the cause of eczema solare and ephelides; the heat of the fire, of the pigment spots on the shins of elderly people. Sweat, again, is a very common irritant, producing the erythema which usually accompanies sudamina and also intertrigo of opposed surfaces. Scratching, as a cause of traumatic dermatitis, has been repeatedly referred to.

III. Febrile Rashes. We must take care never to forget the possibility of a cutaneous eruption being part of an acute exanthem. The use of a clinical thermometer is a great help in this respect. Variola is frequently mistaken for syphilis and other affections.

IV. Other cases are due to certain kinds of food or to drugs. They have been described above.

V. Syphilodermata. When we have satisfied ourselves that the eruption before us is not factitious, nor directly traumatic, nor a symptomatic eruption, we may next consider whether or not it is due to syphilis. In this inquiry it is undesirable to ask questions, the answers to which are as apt to mislead as to guide aright.

1. We should first consider the *color* of the affected skin, remembering, however, that the pigmentation which gives the so-called coppery or raw-ham tint to a syphilitic eruption is the same which is sooner or later produced by all forms of dermatitis. Psoriasis, chronic eczema, lichen planus, and prurigo, may all produce shades which bear the closest resemblance to syphiloderma.

2. The lesions of syphilis are multiform. It is rare in any but syphilitic affections to find mere hyperæmia in one part, and associated pustules, papules, scales, or ulcers, in others; and it is not

often that a syphilitic eruption exhibits only a single elementary lesion.

A pustular eruption in an adult should always suggest the question of syphilis when that of scabies has been answered in the negative.

3. Syphilitic eruptions, for some unknown reason, *do not itch*—the exceptions to this rule are remarkably few; they usually occur during the stage of scabbing of pustular rashes or during the healing of tertiary ulcers. An ordinary secondary syphilide may, however, as a rare exception, be so irritable that wheals and scratch-marks are produced. On the other hand, psoriasis is often free from irritation, while the degree of itching of eczema, and even of scabies and prurigo, varies greatly.

4. The local distribution of syphilitic disease is a great aid in diagnosis. Specific eruptions are certainly not, as a rule, symmetrical; the early roseolous rash is only so because it is general, and therefore, upon a surface like the human body, more or less symmetrical. Moreover, as it chiefly affects the face, chest, and trunk generally, it is near the middle line. But we do not see symmetrical patches of syphilide in corresponding parts of both sides of the face, both sides of the trunk, or the right and left limbs. In all but the earliest syphilides the affected patches are very decidedly and constantly unsymmetrical, irregularly scattered over head, trunk and limbs, and chiefly remarkable for having no well-marked seats of predilection.

The forehead, especially about the roots of the hair, is, however, very frequently the seat both of the early and middle erythematous, scaly, and pustular syphilides, and the palms of the hands and soles of the feet are frequently symmetrically affected with the later scaly eruption.

Practically, when we find a disease of the skin occupying some unusual position we should at least consider the question of syphilitic origin.

5. These signs alone or in combination serve to distinguish early specific roseola from erythema, eczema, scarlatina and measles, and the later eruptions from eczema, lichen, scabies, impetigo, and psoriasis.

The eruptions of *congenital syphilis* which are most liable to be mistaken are: The so-called pemphigus of infants, which is known by its affecting the palms and soles; rupia, which, by the form of the crusts and the ulcerated surface beneath, may always be distinguished from impetigo; an erythematous rash of the nates and genitals of infants, which is distinguished from eczema of the same parts, also common at that age, by its coppery color, its blotchy distribution, and more defined margin.

The *tertiary ulcers* of syphilis are distinguished by their appearing on unusual places, by their punched-out edges, circular or so-called horse-shoe shape, and by their usually producing little pain or discomfort. Tertiary ulcers have no predilection for the outer side of the leg, but inasmuch as the part above the inner angle is, for anatomical causes, the chosen seat of varicose ulcers, most ulcers in the first position will be syphilitic and in the latter not. For the same reason most ulcers on the arms are found to be tertiary.

VI. Tineæ. The next group of skin diseases includes those which are due to vegetable parasites—tinea versicolor of the trunk, eczema marginatum of the perineum and thighs, tinea circinata of the neck and other parts, tinea sycosis of the chin, and tinea tonsurans of the scalp. In all doubtful cases the microscope should be employed.

Tinea of the scalp is rare in adults, and tinea circinata still more so; tinea marginata occurs only in adult males.

VII. Primary Superficial Inflammations. To distinguish the superficial from the deeper kinds of dermatitis, we should notice whether the cutis alone is infiltrated and thickened, or whether it is bound down by adhesions to the subcutaneous tissues. The presence of scars, however slight, is a proof that the process has gone deeper than the papillæ, and has more or less extensively destroyed the papillary layer. Superficial inflammations, excluding those due to the acarus, to pediculi, and to other direct irritants, and excluding also those which are the result of vegetable parasites and of syphilis, fall with respect to their treatment into three large groups:

The first group, represented by impetigo and most forms of eczema, consists of inflammations which are subacute, and accompanied with burning, itching and pain, sometimes with a slight degree of fever.

The second group of superficial inflammations of the skin is typically represented by psoriasis, but includes lichen planus, the more chronic, dry, and obstinate forms of eczema, and true prurigo. These affections are chronic, with little irritation, exudation, pain, or active signs.

The third group is that of erythemata.

VIII. The Acne Group. Acne, both in its pathology and etiology differs from other forms of dermatitis. The age of the patient, and its distribution, are sufficient for diagnosis. It is at once a superficial and a deep dermatitis, and is often followed by scars. Its treatment consists entirely or almost entirely in local applications directed to the correction of the sebaceous affection. With acne may be classed sycosis and furunculus.

IX. Deep Affections. When we have ascertained that the affection of the skin is deep, that is to say, that it goes below the papillary layer, the field of diagnosis is limited.

Excluding erysipelas, which is distinguished by its acute character and febrile symptoms, excluding the pustular affections which affect the skin deeply and produce scars only at isolated points, such as acne, variola and zona, and excluding, thirdly, leprosy and other exotic diseases, we have to distinguish in the great majority of cases which come before us in this country—first, traumatic and varicose ulcers; secondly, gummata and syphilitic ulcers; third, lupus; fourth, rodent ulcer, and carcinoma of the skin.

With regard to the first of these, we must not assume, because a sore upon the skin is said to be the result of a blow or a kick, that it is purely traumatic, for syphilitic ulcers often arise in this way. Malignant ulcers are rare, and are usually obvious from the age of the patient,

the pain they occasion, their tumid margins, and their blood-stained secretions. Moreover, they are, with few exceptions, confined to the neighborhood of the orifices of the body, especially the lower lip, the urethra, the vulva, and the anus. Rodent ulcer, however, is very difficult to be sure of. Its locality, its slow and painless progress, and its belonging to the latter half of life, usually serve to distinguish it from lupus; and its being single, excessively chronic, and unaccompanied by nodes or other syphilitic lesions, are the best characteristics for diagnosis from a tertiary ulcer.

The Subcutaneous Connective Tissue. Swelling of the surface is an indication of change in this tissue. Œdema, myxœdema, subcutaneous emphysema, dystrophies, scleroderma, brawny indurations, and local subcutaneous swellings are the principal ones to be considered.

Œdema; Dropsy. The lymph spaces of the subcutaneous connective tissue become overdistended with serum, causing an accumulation to which the general term *dropsy* is applied. If the accumulation is local and confined to small areas, it is known as *œdema* If it is general, and in addition the large lymph cavities, the pleura, the peritoneum, and the pericardium contain fluid, it is known as *anasarca.* Accumulation occurs because more fluid is poured out by the vessels than can be removed by the lymphatics and veins. This may depend either upon obstruction of the veins and lymphatics or excessive exudation from the bloodvessels or both. The former condition, however, is rare, and usually local, because, unless the obstruction to them is very great, the veins and lymphatics are able to carry away more fluid than is effused from the capillaries. (1) *Local capillary change* from inflammation or the effects of poisons. The change, therefore, as often seen, must be due to some process in the capillaries. Formerly it was thought that this general process was of an inflammatory nature, but at present it is believed to be due to the influence of poisons, probably absorbed from the intestinal canal, modifying and altering the nutrient small vessels. Thus the œdema and general dropsy of albuminuria, particularly in the early stage of that affection, is thought to be due to a common poison circulating in the blood, which also causes the nephritis.

Brunton (to whose article I am indebted for these remarks) states that Mahomed found a pre-albuminuric stage of scarlet fever, in which he noticed a peculiar reaction of the urine, which gave a blue color with guaiac. A brisk purgative when this reaction was noticed would prevent the occurrence of albuminuria; whereas, if the drug was withheld, albuminuria always followed. The purgative removed the poison which caused the nephritis and œdema.. It is well known that in urticaria there is marked local œdema. Brunton thinks that some poisons circulating in the blood cause paralysis of the secreting power of the sweat glands, on account of which there is not only effusion from the bloodvessels, but at the same time such changes in the secreting cells take place as to produce an acid, the local action of which, upon the capillaries, causes a further transudation of fluid. That acids circulating in the blood have the power of creating œdema, the experiments of Cash and Brunton fully

demonstrate. While, therefore, in the œdema of Bright's disease in its earliest stage, and in urticaria, we have this explanation for the phenomena, other factors are causal in other forms of œdema. (2) Œdema will occur whenever there is obstruction to the flow of lymph. This obstruction may be in the lymphatics or the veins. In the former it may occur (*a*) from want of muscular action; (*b*) from want of inspiratory action of the thorax; (*c*) diminution of the diastolic suction of the heart; (*d*) positive pressure on the veins. In the latter, obstruction of the veins is caused by conditions similar to that in the lymphatics, and arises from (*a*) want of muscular action; (*b*) want of movement of the thorax; and (*c*) feeble action of the heart; and in addition it is likely to be caused by (*d*) complete arrest of blood flow from pressure upon the vein from without, or plugging within. It can readily be seen, with a little knowledge of physiology, how the above factors favor the development of the second common form of œdema, namely, that due to disease of the heart and to venous obstruction. The baneful factors are those which retard the flow of blood. Hence, the œdema of passive congestion. (3) A third form of œdema, usually slight, occurs in cases of anæmia. Several factors combine to produce it; (*a*) the watery condition of the blood; (*b*) the condition of the capillaries; and (*c*) vasomotor paresis on account of imperfect nutrition of the vasomotor centres. Finally, (4) œdema may be of nervous origin. The œdema that occurs in diseases or injuries of nerves belongs to this class. Thus far no satisfactory explanation has been given, unless it arises because of alterations in the permeability of the vascular walls.

Mode of Recognition. Whether the accumulation is in the lymph spaces, in local areas, or engorging the entire subcutaneous tissue, the œdema is not difficult of recognition. The part is swollen, the surface is pale, the temperature is usually low, and the affected area pits on pressure. This is more pronounced if pressure with the finger is made over a part which is seated upon a firm background, as bone. Œdema of the ankle or over the tibia is more readily recognized than œdema in the calves.

Œdema is to be distinguished from—(1) Inflammatory swellings, by the absence of the classical signs of inflammation, pain, heat, and redness. (2) Swelling due to myxœdema differs from œdema in the absence of pitting on pressure, the occurrence of induration, which resists the pressure of the finger, and in the occurrence of anæsthesia or analgesia. (3) Swelling due to connective tissue dystrophies are excluded because they are hard localized areas that do not pit on pressure, and that are not seated in dependent parts of the body. They are found on the arm, for instance, or on the leg, or about the flanks and in the axillæ. (4) The subcutaneous swelling due to emphysema differs from the swelling of œdema, in the fact that it arises in the course of some disease of the air-passages and on palpation the crackling sensation of air under the finger is distinctly felt, while there is no pitting on pressure. In the cases that the writer has seen the parts were particularly tender, although pain is said to be absent usually in subcutaneous emphysema.

Diagnostic Significance. The significance of œdema depends upon its location, its mode of development, and its association with disease of other organs or structures of the body.

Situation. The œdema may be local or general.

Local Œdema. *Local*, or at times unilateral, œdema occurs when there is pressure on a vein or occlusion of it by a thrombus. Œdema of the arm, on account of disease of the lymphatic glands in the axilla, and œdema of the leg, on account of thrombosis of the femoral vein, are examples of this form of local œdema. Of marked diagnostic significance is the local œdema that is seen over inflammatory areas. It is an indication of suppuration. It is due to obstruction of the lymph circulation. It is seen over the mastoid when its cells are the seat of inflammation; over the parotid gland under the same circumstances; at the side of the thorax in empyema; over the præcordia in purulent pericarditis; over the surface of the liver in some cases of hepatic abscess; in the abdominal parietes in purulent peritonitis, but more marked at the primary focus of inflammation, as the gall-bladder region or the region of the appendix.

Œdema from the above causes is of local orgin. Œdema from general causes usually develops gradually and begins in special localities. We are accustomed to see it in the course of various affections at first in the *feet* or the *face*.

The Feet. Œdema of the feet or ankles is usually due to disturbance of the circulation, and is a form that arises in heart disease, or in the course of any exhausting and debilitating disease in which the nutrition of the heart has suffered seriously. The organic change (dilatation) which takes place in the heart-muscle in the course of *obstructive valvular disease* and in *lung* disease is attended by œdema, beginning at first in this situation. Later a general dropsy may ensue. But œdema of the feet may occur from another cause, *i. e.*, *anæmia*. In all forms of anæmia puffiness of the ankles is seen. An explanation of the cause has been given. Similar localized œdema occurs in individuals of relaxed fibre, in the evening after a day of considerable physical exertion.

Œdema of the Face. Œdema may begin or remain localized in the face, and is very striking. (See Face and Eyelids) It may be limited to the eyelids, as a simple puffiness, or may spread over the entire face, causing complete obscuration of the normal outlines. The signs of œdema are not different from those of other situations. It is the œdema of *renal* disease, and as a special characteristic differs from œdema of the feet in that it disappears toward night and is more marked in the morning on rising. Of all forms of local œdema it is the most grave, and should at once command attention to the condition of the urine, particularly if the patient has just had an attack of scarlatina, or if it occur in a female who is pregnant.

The Arms and Thorax. Another form of local œdema, bilateral in situation, occurs when there is internal pressure within the thorax on account of aneurism, or disease of the mediastinal glands. The œdema is then limited to the arms, or to the arms, head, neck, and thorax. Such œdema is usually associated with cyanosis of the hands and arms. There is also marked distention of the veins of the upper part of the body. The œdema has been found, in a few instances, to be more marked on one side than the other. This has occurred in cases of aneurism in which the sac of the aorta communicated with the

vena cava. Either the collateral circulation on one side had been established, or pressure was greater on the left innominate vein. The œdema is sometimes limited to the head and arms. If the obstruction of the superior cava was situated below the entrance of the azygos vein the chest shared in the venous congestion and resulting œdema. If, on the other hand, the obstruction was above the azygos vein there was no œdema of the chest wall. Œdema of this character is usually easily recognized, because of the symptoms above indicated, with other pressure symptoms due to disease of the mediastinum and with the results of physical examination, which reveals the presence of a tumor in the thorax. The above-described œdema usually develops slowly, hand-in-hand with the other symptoms. At times, however, it occurs suddenly. *Sudden œdema* in this situation is always due to an aneurism, which has ruptured into the vena cava (see above). The sudden onset is attended by physical signs of aneurism, or, if they are not present, by a murmur characteristic of the communication between an artery and a vein. It must be confessed that often the physical signs are not precise and the murmur is absent. The suddenness of the peculiar localized œdema is the chief point of diagnosis in favor of this rare form of aneurism.

The Œdema of Trichinosis. (See Face.) In addition to the face (which see), œdema of the skin over the affected muscles is common. This œdema occurs early in the disease, disappears after a few days, to return again later. It is localized in the muscles, and is associated with the growth of trichinæ in them. It is distinguished from cardiac and renal dropsy by the above means and by the fact that the scrotum and labia majora are never œdematous.

General Œdema. Œdema of the face and feet may become general. In cases in which the face is first œdematous, its extension may be very rapid, so that twenty-four to forty-eight hours after the swelling is noticed the whole body is in a state of anasarca. The extension of œdema which primarily was seated in the feet and legs (cardiac dropsy), throughout the rest of the body is more gradual, and develops along with signs and symptoms indicating weakness of the heart. As it advances, in contrast with the œdema of Bright's disease, the hue of the patient changes. Cyanosis gradually appears. This may be seen first in the extremities, which are also œdematous. Finally the face and lips take on this peculiar hue. On the other hand, in the general anasarca that follows the local œdema of the face, of Bright's disease, pallor occurs, and as the œdema increases it becomes more and more of a waxy hue. With the waxy hue of the face, the extremities become glistening or shining in appearance.

Angio-neurotic Œdema. This curious affection is not of frequent occurrence. It may be present in the individuals of several generations of a family. The attack comes on suddenly. The swelling is circumscribed. It may appear on the face, on the brow, the lips or cheek. The eyelid is a common situation. It may also occur on the backs of the hands, the legs, or in the throat. It remains but a short time, and disappears as quickly as it came on. The outbreaks have exhibited distinct periodicity. Local symptoms of itching, heat, or redness, or

general urticaria may precede the swelling. The sudden swelling causes great deformity. If the upper lip is affected the mouth cannot be opened; if the hands, the fingers cannot be bent. In the hereditary cases the attack recurs every three or four weeks. The danger to life is from œdema of the larynx, which caused death in two of Osler's cases. The general symptoms that attend the attack are gastro-intestinal. Nausea and vomiting, followed by severe colic, occur.

It must not be confounded with simple urticaria, or the giant form of that affection, with which it may, however, have close affinities. It has no relation to arthritic affections. It is regarded by Quincke as a vasomotor neurosis, on account of which the permeability of the vessels is impaired.

RECAPITULATION. From what has been said above the student will observe that œdema may be local or general; that local œdema may be unilateral or bilateral; that œdema may further be subdivided, in accordance with the cause, into inflammatory dropsy, œdema or dropsy of passive congestion, hydræmic dropsy, and vasomotor dropsy. The forms of passive dropsies just indicated may be subdivided into cardiac dropsy, hepatic dropsy, and renal dropsy, dependent on the anatomical cause for the dropsy.

While the account of œdema just given refers more particularly to the subcutaneous accumulation of serum, the same pathology and ætiology apply to accumulations in the large lymph cavities, and hence in addition to general œdema we may have *ascites*, *hydro-pericardium*, *hydrothorax*, *hydrocele*, and *effusion* in the joints. The method of recognition of dropsy of the larger cavities will be deferred until diseases associated with these particular regions are discussed. It must be remembered that œdema or accumulations of serum in cavities may be of local or general origin.

It must not be forgotten that two or more causes may combine to form a dropsy, or that a dropsy of one cause may for a time be dependent upon a second and even a more pronounced factor later on in the development of the disease. Thus (*a*) the dropsy of an hydræmia may be aggravated by that of (*b*) a weak heart which arises from anæmia, to which may be added later the dropsy of vasomotor paresis. The dropsy of Bright's disease is due to (*a*) capillary changes produced by a poison circulating in the blood, and (*b*) later, to the condition of the heart, which frequently undergoes dilatation.

Myxœdema. Swelling of the surface of the body, local or general, is also seen in myxœdema, a condition which simulates dropsy, the distinction from which has been referred to above. In myxœdema the swelling is general. The face is involved. The arms are more markedly swollen, however, than the fingers; the legs more than the feet. The swelling is due to the infiltration of mucin into the connective tissue, and arises from some affection of the thyroid gland. The gland is absent, functionally or actually. The hard, indurated, non-pitting swelling is associated with striking change in the appearance of the face, particularly the nose and forehead. The nose becomes thickened, the forehead more prominent and overhanging. The outline of the face is rounded, so that the term "full-moon" is applied to it. The skin is thickened,

dry, and rough, somewhat translucent in appearance, of a doughy consistence, with a moderate degree of elasticity. The perspiration is diminished. The hands change in shape, they become square or spade-shaped, and the fingers clubbed. The appendages of the skin change. The nails become brittle and distorted, the hair dry, harsh, and brittle, and it may fall out. With these remarkable changes in the exterior, marked nervous and mental symptoms arise. Speech is thick and hesitating, the memory feeble. The intellect is dull and irresponsive, the temper irritable. Sensibility is impaired, particularly in the loss of sensation to pain. Patients have been burned without their knowledge. This occurred to considerable depth of tissue in one of the writer's cases. Abnormal sensations of heat and chilliness are complained of, as well as other paræsthesias. The patient is anæmic, the temperature is subnormal, the heart's action weak, the respiration sluggish. Breathlessness on slight exertion is pronounced, and exertion itself is very difficult, while there is greater sense of fatigue than the exertion and the condition of the organs would warrant. The muscularity is enfeebled. There is impairment of appetite, indigestion, and flatulency. The urine may become albuminous, but for a long time is not characteristic save in amount and specific gravity. The former is increased, the latter lowered.

As the case advances mental and physical failure becomes more pronounced, the patient is subject to hallucinations, and is extremely irritable. Stupor sets in; death may take place in coma, or from uræmia.

Subcutaneous Emphysema. Enlargement of or swelling of the surface, either local or general, may occur on account of air underneath the skin. The primary seat of the swelling is in close proximity to the air passages, and occurs because of communication between them and the subcutaneous connective tissue. In ulcerations of the upper passages, as the larynx or trachea, it may occur, and in rupture of the lungs and pleura in cases where the latter is adherent, air will pass from the former into the connective tissue. From the seat of rupture, or in close proximity to it, as the starting-point, the swelling gradually spreads over the entire body. In a case of laryngeal phthisis under the writer's care it encircled the neck and spread uniformly over the anterior and posterior portion of the thorax. From thence it extended downward until it met a corresponding infiltration of the lymph spaces in the thighs due to serum. The distinction between œdematous swelling and subcutaneous emphysema could thus be made: the latter offered no resistance, did not pit on pressure, crackled under the finger, and was quite tender on pressure. Spontaneous pain was not present, but the weight of the body, in any position assumed pressing upon the part, caused pain.

Connective-tissue Dystrophies. Swelling of the surface of the skin in irregular areas from changes in the connective-tissue are also seen in the so-called dystrophies. The dystrophy is usually due to a localized anomalous overgrowth of connective tissue probably of trophic origin. It can easily be distinguished from œdema by absence of the signs of œdema, or from local inflammatory swelling, by the absence of pain,

heat and redness. The swelling occurs on the arms and legs, usually on the outer aspects, and may occur in various portions of the trunk. In one of the writer's cases the swellings were periodical; or rather, the persistent swellings increased in size at irregular intervals.

Dercum and Henry have described them in cases in which the enlargement was thought by others to have been due to accumulations of fat. The patients presented marked subjective nervous phenomena, paræsthesias of all kinds, with flushings and sensations of sinking and depression. There were areas of anæsthesia, pain and tenderness in the nerve trunks. Pain preceded the advent of the swellings.

Herpes zoster occurred in Dercum's case, and other symptoms of neuritis were marked. The irregularity of distribution of the swellings, their character and mode of development, the recurrence of neuritis and the absence of perspiration distinguished this affection from lipomatosis or excess of fat. The patients were of a neurotic type, and usually mental impairment resulted in the course of the disease. The general nutrition failed, particularly as gastro-intestinal disorders ensued.

Scleroderma. Scleroderma is a hyperplasia of the sub-connective tissue in which there is swelling with induration of the part. It is brawny. The term "hide-bound" is applied to this condition of the connective tissue and skin, on account of which the parts are almost immovable. There is marked stiffness and also pain.

In localized scleroderma the skin has a waxy or dead-white appearance, is brawny and inelastic. There may be preliminary hyperæmia of the skin. Subsequently pigmentation of the hyperæmic area takes place, causing changes in color. Or the pigment may atrophy, causing *leucoderma*. The secretion of sweat is diminished, or entirely abolished. In the diffuse form the affection begins on the extremities or face, accompanied by a sense of stiffness or tension; the skin is unusually hard and firm, and gradually a diffuse, brawny induration develops. The skin cannot be picked up in folds. It may appear normal, but is generally very smooth, glossy, and dryer than usual. Scleroderma may be confined to a limb, or become universal. The appearance of the face is characteristic. It is expressionless, and the lips cannot be moved, while mastication is impossible; then the hands become fixed and the fingers immobile on account of induration about the joints. It is thought to be due to a tropho-neurosis, or to fibrosis of the arteries of the skin, with connective-tissue overgrowth in the adjacent areas.

Brawny Induration. Œdema must not be confounded with the brawny induration of the calves of the legs that is seen in scurvy, which is probably due to deep-seated hemorrhage, or to the affections between the periosteum and the bone which form chronic nodes. It must be remembered, however, that œdema of the ankles is very common in this affection. In a patient recently in the Presbyterian Hospital under the writer's care a brawny induration of the thigh, with painless swelling and stiffness of the leg, appeared to be due to syphilis. It disappeared rapidly under the use of iodide of potassium. The patient was syphilitic.

Subcutaneous Nodules Distinctly Localized. SARCOMATA. The subcutaneous nodules seen in these affections are rarely, if ever, confounded with œdema or other swellings. In sarcoma the subcutaneous tumor becomes attached to the skin, and may change its color. It is usually secondary to sarcoma in some other organ of the body. When primary or secondary to organs in which there is normal pigmentation, as the eye, they become blue or bluish-black. On palpation the surface is rough and uneven if the tumors are in great number.

Primary melanotic sarcomata of the skin can always be distinguished by their color. In both forms of sarcomata the general symptoms of this affection daily become more and more pronounced, and subcutaneous hemorrhages are commonly associated with the local phenomena.

CYSTICERCUS CELLULOSÆ. The nature of the subcutaneous nodules of cysticercus are recognized by microscopic examination. They are usually associated with the larvæ in other tissues, hence the patient will complain of great soreness and stiffness, and may on account thereof be helpless. In a case reported by Osler there was so much numbness and tingling in the extremities with general weakness that the patient was thought to have peripheral neuritis.

RHEUMATIC NODULES. Subcutaneous nodules are seen in rheumatism or in patients who have had frequent attacks of that disease. They are common in the young. They are particularly frequent in cases of rheumatic endocarditis. They may occur in large numbers, and vary from a small shot to a large pea. They are of fibrous structure. They are attached to the tendons and fasciæ, particularly on the fingers, hands, and wrists, but may be found on the elbows, knees, the scapula, and the spines of the vertebræ. They may occur independently of an attack of rheumatism, or follow in its decline.

The Temperature. Fever. In conditions of health the body temperature is maintained constantly at about 98.6° F. (37° C.). This stability of temperature is due to the central regulating apparatus called the thermotaxic mechanism, which controls the production and the dissipation of heat. Fever is a condition characterized by an increase of temperature with, usually, increased disintegration of nitrogenous tissue. The muscles and large glands, as is well known, are the chief seat of heat production. Both heat production and heat dissipation are believed to be under the control of the nervous system, either through the motor nerves or special nerves which pass with them to and from definite centres in the brain called heat centres. In conditions of disease this thermotaxic mechanism may be altered so that the normal temperature is increased or lessened. (1) There may be elevation of temperature from diminished dissipation of heat, though not necessarily increased nitrogenous disintegration and disordered function. Or, (2) there may be increased production of heat with diminished dissipation, hence the temperature will naturally be higher than if the increased heat production were accompanied by normal heat dissipation. (3) There may be increased heat production and at the same time increased heat dissipation, in which case there would be the increased waste of fever with or

without any elevation of temperature. (4) It is possible that heat dissipation may be greater than heat production, or that the thermotaxic mechanism may be disturbed so as to promote loss, in which case there will be subnormal temperature.

Mode of Determination of Fever. The temperature of the body can be roughly estimated by the hand of the physician, but this method is open to many sources of error. The skin is at times hot and gives a deceptive sensation of considerable elevation of temperature, whereas when tested by the thermometer the temperature is but slightly or not at all above normal. So, too, when the skin feels cold and clammy in phthisis and during a chill from any cause, the actual temperature of the body is decidedly above normal, and may be as high as 103° or 104°. To insure accuracy, therefore, it is now almost the universal custom to employ clinical thermometers. They are of a convenient size and shape for insertion under the arm or into the mouth, rectum or vagina. The better ones are provided with an indestructible index, so that the mercury in the capillary tube remains stationary at the highest level to which it rose when the thermometer was in the mouth or axilla. When not provided with such an index the reading must be made when the thermometer is still in position.

Thermometers vary in the accuracy with which they register temperature. The best ones are compared with an acknowledged standard and sold accompanied by a slip of paper which gives their fractional variations from the standard. When the exact temperature is a matter of great importance it should be taken in the *rectum* or *vagina*, as their temperature is more nearly that of the body. It is of advantage to take the temperature in the rectum in children or in patients who are comatose. This situation is also a good one to select when a bath is being administered. If possible, scybalous masses should be removed from the rectum. At least an incorrect reading may be obtained if the thermometer should happen to be plunged into the fæces: this must be guarded against. From motives of delicacy, however, the axilla is to be preferred to the rectum and vagina on all ordinary occasions. The temperature it records is somewhat less than a degree below that of the rectum. The temperature of the *mouth* is above that of the axilla and below that of the rectum. It has some advantages over that of the axilla, being more accessible and recording the temperature more quickly and more accurately. Nevertheless, as the physician's thermometer is carried from patient to patient, some place of taking the temperature should be selected which is less capable than the mouth of absorbing disease germs. The *axilla* is, therefore, by common consent the usual place of taking the temperature. Observe two precautions. (1) Before introducing the thermometer see that there is no undue moisture; if there is, the axilla should be wiped dry, otherwise a lower than the true reading will be obtained. (2) See that the instrument is inserted into the armpit and does not project beyond the posterior fold, and that it is not caught in a fold of the undershirt or night-dress. After the thermometer is in position the arm should be brought gently across the chest and kept in that position until the instrument is withdrawn. The arm should not be held rigidly, as such

muscular action increases the hollow of the armpit and may keep the sides apart, instead of in contact, as they should be to make a correct reading. The length of time required to take the axillary temperature will depend upon the instrument used; generally from five to eight minutes are required. Some very delicate thermometers register in one minute, but they are too fragile for ordinary use. If the index is in such a position that it can be seen it is proper to withdraw the thermometer when the mercury has ceased to rise for two minutes.

The index, of course, must be shaken down to normal or slightly below normal before the thermometer is ready for use; and the instrument must be carefully cleansed after use. These are hints which only students need to be reminded of.

In children who are restless the temperature may be taken in the groin, as the folds of fat readily admit of completely enveloping the bulb of the thermometer. The height to which the mercury rises will correspond to the temperature in the axilla. The temperature of the *urine* corresponds exactly to that of the body if taken when freshly passed and during the act in males, before being received into a receptacle. Sometimes this method of securing the temperature is resorted to, particularly in patients who may act as malingerers and in whom it is desirable to have the temperature taken in the physician's presence.

If the *mouth* is selected as the place in which the temperature is to be taken, care should be exercised that the thermometer is placed underneath the tongue, or along its side between it and the lower jaw, and retained in position by the lips of the patient. If the teeth are set firmly on the thermometer it may be broken, or, what is of still greater importance, it will be tilted out of position and a correct reading will not be obtained. The lips should be closed and breathing be carried on through the nostrils. Four to seven minutes is sufficient time to allow it to remain in position. The patient should not have taken ice or anything cold prior to the observation.

Observations of the temperature should be made twice a day, in the morning and evening, and, as far as possible, at the same time on successive days. It is frequently desirable to have the temperature taken every two or three hours, and sometimes at more frequent intervals.

In obscure cases the observations should be repeated at night as well as during the day. In this manner the presence of unsuspected tuberculosis may be revealed, or the occurrence of suppuration in some portion of the body, the seat of an obscure process, definitely determined. It should not be forgotten, however, that the temperature may be taken too frequently for the patient's good, the disturbance of his needed rest being distinctly harmful.

As the general range of temperature and its diurnal variations are of more importance than the absolute temperature at any one time, thermometers not perfectly accurate in their reading are still good enough for clinical and therapeutic purposes.

Physiological Variations of Temperature. The temperature is subject to *physiological variations*. 1. It rises from seven or eight in the morning until seven or eight in the evening, at which time

it reaches its maximum. It then begins slowly to fall, reaching its lowest point in the early hours of the morning, between two and four. This *diurnal fluctuation* does not usually amount to more than a degree. 2. *Exercise*, etc. Violent exertion raises the temperature, and so does a heated atmosphere, cold having a contrary effect. 3. *Age*. In infants and young children, up to puberty, the temperature has a somewhat higher range, and is subject to greater variations than at a later period. In very old persons the temperature may be subnormal. The *normal* axillary temperature of adults is 98.6°F. The period in the twenty-four hours in which the temperature is at its lowest ebb is from 12 P.M. to 4 A.M. It may then be subnormal. The writer has known an over-cautious parent have this physiological fall made the subject of meddlesome observation and ill-judged treatment.

PATHOLOGICAL VARIATIONS OF TEMPERATURE. An elevation of temperature above the normal not to be accounted for by external heat or severe exhaustion may be considered febrile, and is pathological. The range of febrile temperature varies from above normal to 105° or 106° in ordinary cases. A range above 106° may occur, but is not usually compatible with life. Certain terms have been applied to various degrees of temperature to indicate in a general way the degree of fever.

Below	35° Cent. = 95.0° Fahr.		Very low, or collapse temperature.
	36 = 96.8		
About normal	36½ Cent. = 97.7° Fahr.		Subnormal temperature.
	37 = 98.6		Normal temperature.
About	37½ Cent. = 99.5° Fahr.		Slightly above normal, or sub-febrile temperatures.
	38 = 100.4		
	38½ = 101.3		
About	39 Cent. = 102.2° Fahr.		Moderately febrile temperature.
	39½ = 103.1		
About	40 Cent. = 104.0° Fahr.		Highly febrile temperature.
	40½ = 104.9		
Above	41 Cent. = 105.8° Fahr.		Hyperpyretic temperature.

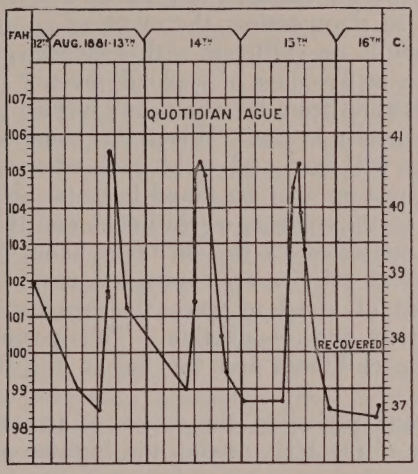
(From FINLAYSON.)

THE DEGREE OF DANGER. In general the danger to the patient increases with the height of the fever, but the duration of the high fever modifies this greatly. A temperature of 106° on the second or third day of an acute lobar pneumonia is not rare, such cases frequently ending in recovery, while a temperature of 105° in the second or third week of typhoid fever is of much graver significance. Da Costa has reported a case of cerebral rheumatism in which the axillary temperature reached 110°, yet the patient recovered. In the case of injury of the spine reported by Teale the extraordinary temperature of 122° was recorded, and the temperature range for days was between 112° and 114°. The patient recovered.

THE TYPES OF FEVER. Fevers are divided in accordance with the character of their range into certain definite types. The types may be indicative of special processes. It is certain that the recognition of a particular type forms a positive aid to diagnosis. The fever that continues for more than two days, in which the difference between the daily maximum and minimum of temperature is less than 2° is known as

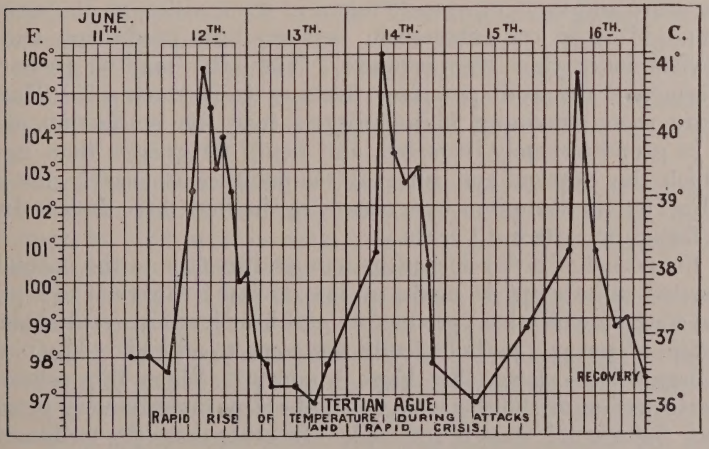
continued fever. (See Fig. 8.) The fever existing more than two days, in which the daily difference is greater than 2° is known as *remittent* fever. Further, a fever in which there is a rise of temperature followed by a fall to or below the normal, occurring at least once during

FIG. 3.



the twenty-four hours, is known as an *intermittent* fever. The paroxysms may occur daily every second or third day, or once a week. When the paroxysms occur daily the intermittent is of quotidian type

FIG. 4.



(see Figs. 3 and 5); every second day, tertian type, one day intervening without fever (see Fig. 4); every third day, quartan type, two apyretic days intervening.

The Course of the Fever. Fevers have frequently a definite course, known as (1) the initial stage; (2) the fastigium; (3) the period of defervescence. During the *initial* stage the temperature rises higher each hour, or if extended over days, each day, than the preceding hour or day—in this latter instance interrupted by the daily fluctuations. The stage may last from a few hours, as in a paroxysm of intermittent fever, to four or five days, as in typhoid fever. In this stage we have a chill such as characterizes the onset of an intermittent fever, or the recurrent chills or chilliness with headache and backache that attend the first four or five days of typhoid fever. During this stage, also, the heat dissipation from the cutaneous surface is diminished, and heat dissipation generally is less. When the hand is placed upon the patient the surface will be found to be cool, whereas the temperature in the mouth or rectum will be found to be far above normal. The patient complains of the coldness or chilliness, and the low temperature of the surface is indicated by the shrunken hand, the pallid, pinched face. The peripheral arteries are contracted, and hence cause diminution in the amount of blood to warm the skin and to compensate for the loss by radiation and conduction. This peripheral contraction is the cause of the chilliness and the fall in the temperature of the skin.

During the second period of the course of pyrexia—the *fastigium*—the temperature of the body attains the highest point, and remains almost stationary, or may vary but a degree or two between maximum and minimum. It may last a few hours or from two days to three or more weeks, during which time it may oscillate to the maximum point of the first day. The temperature of the surface of the body is about the same as that of the deep parts, particularly in cases of pneumonia, measles, and scarlet fever. In typhoid fever, acute rheumatism, and phthisis, during this period, there may be a difference in the external temperature and the temperature taken in the cavities, as the mouth or rectum. More or less antagonism between heat production and heat loss exists under these circumstances. The latter may be greater than the former, if the skin perspires freely as in rheumatism. The temperature then remaining high indicates that the production of heat must be proportionately increased, and hence far greater than in cases in which the external and internal temperature are nearly the same. (See Fig. 6: the fastigium here occurs in the first three days. In Fig. 9 the fastigium lasts until crisis.)

In the period of *defervescence* the temperature falls to the normal. In this period an attempt is made by the economy to return to a physiological state, in which heat production and heat loss are evenly balanced. The state of pyrexia pathologically has come to an end. The termination may be by *crisis*. (See Figs. 4 and 9.) When this takes place the perturbation of the thermotaxic mechanism must be very great, but at once the normal state is resumed. In other cases the termination is by *lysis*—the temperature falls a degree or two each day until the normal is reached. (See chart of Typhoid Fever.) It seems that the thermotaxic mechanism of health is restored with difficulty. In some cases, in the period of defervescence the aberrations are sometimes very

remarkable. It seems as if the thermotaxic mechanism which controls heat loss was in a convulsive state. The temperature rises and falls irregularly, gradually assuming the normal position only as the strength of the patient increases.

THE MODE OF ONSET OF THE INITIAL STAGE. The onset may be sudden or gradual. 1. The sudden onset occurs in acute diseases, as tonsillitis, pneumonia, and gastro-intestinal disorders of children, in erysipelas and in intermittent fever. Within a few hours the maximum of temperature is reached. (See Fig. 9.) 2. The mode of onset may be gradual. The initial stage is prolonged under these circumstances, as in cases of typhoid fever. (See chart of Typhoid Fever.)

THE MODE OF DECLINE IN THE PERIOD OF DEFERVESCENCE. A sudden fall of temperature at the termination of a disease is known as crisis, which is also characterized by copious perspiration, a "critical sweat," or by the passage of a large quantity of urine, and sometimes by several large liquid stools. The pulse rate and respirations fall correspondingly with the temperature. (See Fig. 9.)

The defervescence may, however, occupy several days, in which case it is said to occur by lysis. In this case the sweating is less marked, but may occur through several days. The slowing of the pulse and respiration likewise occur more gradually. (See chart of Typhoid Fever.)

Diseases that are of sudden onset usually terminate with sudden decline, and correspondingly, in diseases in which the onset is prolonged the decline is also prolonged. Many cases in which the natural termination is by crisis may terminate by lysis. This change is usually due to complication. (See Fig. 6.) In measles, pneumonia is usually the causal complication, while in pneumonia it is empyema or endocarditis.

THE DAILY RANGE OF THE PROLONGED INITIAL STAGE, AND THE FASTIGIUM. The daily range of the temperature in fever generally corresponds to the normal variations. That is, the temperature is lower in the evening than in the morning. The difference in the daily range varies in the different types of fever—generally, as previously noted, the continued fevers having a smaller difference between morning and evening temperature, the intermitting fevers a larger difference between the two.

Sometimes there is *inversion* of the normal range. The evening temperature is lower than the morning; although a rare condition, this is of serious import. It is seen in the course of typhoid fever in the more severe cases, and occasionally in tuberculosis.

RECRUDESCENCE. After the temperature falls to the normal, in many cases fever is again renewed. This may occur from a number of causes. It may be from perturbation of the nervous system on account of excitement, over-exertion, and the loss of sleep, or from indigestion. Slight aberrations, which in health would not modify the temperature, in illness cause pronounced oscillations. Recrudescence, further, may be produced by a relapse. After the afebrile period following typhoid fever, for instance, the temperature may rise and a full recurrence of the disease take place. (This occurrence is well seen in the temperature charts accompanying the article on Typhoid Fever.)

The Symptoms of Fever. Pyrexia, or increased temperature, is not the only evidence of fever. The production of heat within the body is not alone due to increased tissue change. It may be due to increased oxidation of sugar, for instance, which is part of the substance of the body. Physiologists have found that a high temperature may take place, and yet the quantity of urea and of carbonic acid which is discharged may not be as great as that discharged by a healthy person who is taking active exercise or who has eaten a large meal. It must be remembered, therefore, that it is not heat production alone, but *alterations of heat regulation*, which cause pyrexia and its phenomena.

Wasting. Wasting of the body is a striking feature of fever. There is no doubt that even in fever of moderate duration great wasting of the solid structures takes place. At the same time the blood wastes (see observations of Thayer), and the various fluids of the body are also diminished; hence the disorders due to diminished secretion of glands are prominent in the course of fever. Thirst, diminution of secretions in the gastro-intestinal tract, on account of which loss of appetite and indigestion and constipation arise, all indicate the wasting of the fluids. Scanty urine of high color and specific gravity is due to the same cause.

The Pulse Rate. Acceleration of the pulse is one of the phenomena that attend pyrexia. While the increased pulse is in all probability a result of the increase in temperature, and is the usual occurrence, other circumstances will cause a change in the pulse rate in fever patients. Thus, in basilar meningitis, although there may be a high fever, the pulse is not increased. On the other hand, some cases which usually give rise to fever, as diphtheria and peritonitis, may be afebrile, and yet the pulse is very much accelerated.

While there is acceleration of the heart's action, the rapidity with which the blood flows in fever and the arterial tension do not bear a due proportion to the acceleration. The true febrile pulse is not dicrotic. In the early stages of fever the pulse is large and hard, the arterial tension is high, and the vessels full. In the later stages arterial relaxation takes place, and with low pressure the pulse becomes soft and feeble, and often small. The pulse is rapid, and dicrotism, or even hyper-dicrotism now becomes a prominent feature. The heart beating rapidly empties itself incompletely and discharges less rather than more blood into the arteries. The impairment of the cardiac beats is no doubt due to the degenerations which are liable to take place on account of the high temperature, and is not dependent upon any special febrile affection. Such changes also take place in the glands, particularly the liver and kidneys, and are known as parenchymatous degenerations or cloudy swelling. On account of these changes in the cardiac muscle, in the later stages of fever, thrombi may develop, and death takes place from heart-clot.

Respiration Increased. The respirations are increased in fever, probably because of the close dependence of the regulating centres of respiration on that of the heart. The heated blood acts as a stimulant to the respiratory centre. As proof of this the hurried respiration of pneumonia ceases as soon as the temperature falls, notwithstanding the affected part of the lung remains hepatized.

Cerebral Symptoms. Delirium and other nervous symptoms may attend fever. They are not dependent upon the increased temperature of the blood alone. No relation appears to exist between the intensity of the fever and the severity of the delirium. In relapsing fever a temperature of 106° occurs with the mind clear. In certain cases of typhoid fever a temperature of 103° is attended with marked delirium.

If fever persists for a short time, a low asthenic state may develop. Because the symptoms resemble those of typhus fever, the term typhoid is applied to them, and the condition about to be described has been known as the *typhoid state.* The expression is dull and heavy, the capillaries of the face are congested. There is stupor and sluggishness of mental processes, so that the patient is slow in answering questions. The stupor is attended with low muttering delirium, and may be followed by complete unconsciousness. The pupils are contracted, the eye heavy and dull. The patient is so prostrated that he slips down into the bed from the pillow. There is marked subsultus tendinum. The tongue, if protruded, comes out slowly and is tremulous. It is dry and brown, and the mouth and teeth are covered with sordes. The sensibilities are blunted so that food and drink are not asked for, or particularly relished if given. Involuntary discharges take place from the rectum and bladder, and the incontinence from retention of the urine arises. The pulse is small, feeble, and dicrotic, the heart sounds are weak and feeble. The first sound become short and snappy like the second, or may be absent entirely. Venous stases take place in the dependent portions, particularly in the back of the lungs. As œdema or hypostatic congestion advances the breathing becomes shorter and labored. More or less cyanosis then creeps over the general surface. The urine becomes more and more scanty and high-colored, contains albumin, and there may be some blood.

The typhoid state may continue for many days, or even last two or three weeks, although not in so advanced a degree as has just been described. It is more likely to supervene when there is excessively high temperature, but it also occurs in the course of an illness with prolonged temperature of moderate degree—that is, of 103° F. Although in all probability it is due to the direct effects of heat upon the nerve centres and the organs of the body, yet there are cases in which the temperature is not high, and yet all the symptoms of the typhoid state supervene. While the typhoid state is common to *typhoid fever*, it occurs also in *pneumonia* and *septicæmia*, and may be seen in most typical form in other conditions in which fever is not a pronounced symptom; thus, in *uræmia*, in the later stages of *softening of the brain*, in *paresis*, or in allied nervous diseases, the symptoms of the typhoid state are most striking. In this class of cases it certainly cannot be attributed to the fever, and in all probability is due to the depressing effect on the nervous system of material which should be excreted from the body, a view which has been advocated by Murchison, Flint, and others.

Ataxia, or the *ataxic state*, in fever is a condition the opposite of the adynamic, or typhoid state. In the latter there is weakness, while in the former there is exhibition of strength. In the latter the nerve centres and the vital processes are depressed; in the former they are

stimulated. Ataxia as an exhibition of strength is characterized by a strong pulse, by active violent delirium, so that it is almost impossible to keep the patient in bed; by evidence of great muscular strength. The face is flushed, color bright red, the eyes injected, bright, and active. The tongue is furred, but is not necessarily dry or brown. The delirium may be constant or be pronounced at intervals, and is often maniacal in character. The temperature of the body is high, and a sensation of intense heat when the hand is placed on the skin of the trunk is given off. The patient may complain of a bursting, intense headache. If the ataxic state is not controlled after a few days, or at the most a week, the patient becomes exhausted and lapses into stupor, which may proceed to coma. In some forms, particularly in children, convulsions may take place with excessively high temperature followed by coma. The same exhibition of strength is shown. The ataxia is seen notably in scarlet fever, "cerebral" pneumonia, and forms of typhoid fever. The peculiar behavior of the temperature and nervous symptoms in this affection and in apex pneumonia, or so-called pneumonia of the cerebral type, have led observers to mistake cases for those of actual cerebral disease. Frequently such cases have been admitted into insane asylums for supposed mania. Cases of this character have not been appreciated, an insufficient force of nurses being placed over them, with the result that in a number of instances the patients have jumped from windows or escaped from the room and gone out on the streets.

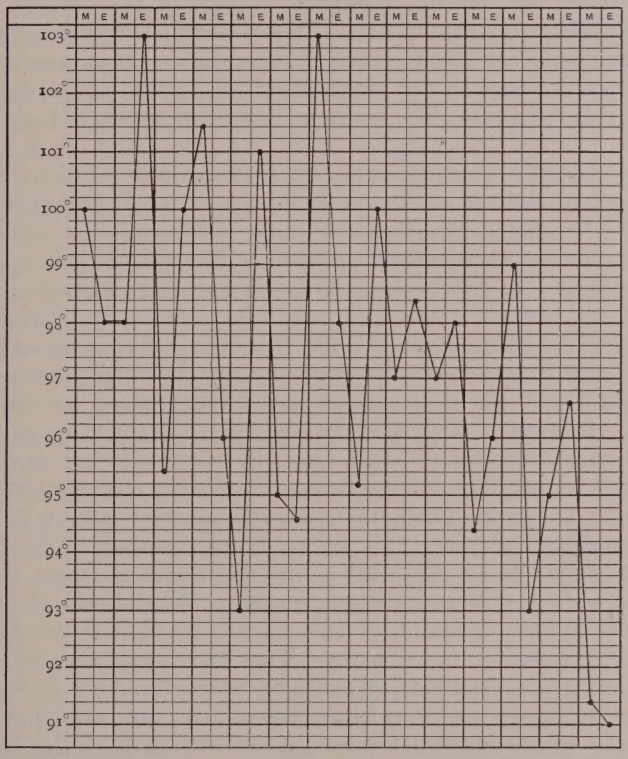
Just as in the adynamic cases it is difficult to determine the exact cause of the extreme perturbation of the nervous system, in *febrile* ataxia it is easy to say that it is due to a high temperature acting on nerve centres; but on the other hand, it is just as easy to say that it is due to a poison, a toxin from infection, which has created the temperature on account of which the nervous symptoms have ensued.

In addition to the increase of temperature as registered by the thermometer, the presence of fever may also be recognized by *flushing* of the face. This may be general or local. The local flush of phthisis and of pneumonia have previously been referred to. *Sweating* is a condition habitual in some fevers. It may occur throughout the course of the disease or at certain stages only in the course of tuberculosis, as the early morning or night sweats testify. In such cases it is cold and clammy. The same sweatings are common in the fever of deep-seated suppuration and in disease of the bones. Sweating in defervescence marks the occurrence of crisis. *Dryness* and pungency of the skin more commonly, however, occur in fever. In former times the sense of heat was given different attributes which were said to be distinctive of various affections. Thus the sensation to the hand of the heat in typhus fever was said to be peculiar and characteristic. The degree of fever was also indicated by touch. The thermometer has now displaced this method of reckoning temperature.

Headache and Pain in the Back occur in the acute specific fevers in the initial stage. One or both are nearly always present, but in their most pronounced form in different affections they have diagnostic significance. Thus pain in the back is more pronounced in tonsillitis and smallpox, severe headache in cerebro-spinal meningitis, and protracted throbbing headache in typhoid fever.

Subnormal Temperature. A temperature below the normal may occur independently of fever, but it usually follows the occurrence of pyrexia. It occurs independently in the course of wasting diseases, as cancer, in starvation, at times in anæmia. It is seen habitually in myxœdema and occasionally in diabetes. In certain forms of tuberculosis it is seen to extend over a long period of time, as in tuberculous peritonitis. (See chart under Tubercular Peritonitis.) Sometimes the subnormal temperature may occur suddenly, to be followed by a subsequent increase in the temperature range. Sudden fall of temperature

FIG. 5.



Subnormal temperature. Oscillations in hepatic intermitting fever with jaundice. Catarrh of ducts, with diffused hepatitis. G. W., aged 60. Philadelphia Hospital, 1877.

below the normal may occur in shock, or from hemorrhage from any cause. It may take place from disturbance of the central nerve centres, as from apoplexy, thrombosis, or embolism of the brain; either from shock or from disturbance of the thermotaxic mechanism. It is characteristic in cholera. In the course of organic heart disease sudden pulmonary embolism is also attended by fall of temperature below the normal. In many of these instances the temperature will rise (reaction) after the shock, if the latter is not too profound. This is notably so in apoplexy and in the other conditions indicated in which the presence of an

embolus or thrombus, or the local condition on account of which hemorrhage took place (softening) may act as a source of irritation. In apoplexy the rise in temperature will occur either from central disturbance of the thermic mechanism or from secondary inflammation about the clot. The subnormal temperature that occurs in the course of fever may be due to an accident or complication, as hemorrhage in disease of the lungs, or in typhoid fever, or the sudden occurrence of perforation of the intestine in the latter condition. At the usual period of the termination of acute disease it attends the crisis. More or less collapse usually attends the pathological fall of temperature below the normal. While such fall is the result of accident in many of the diseases mentioned, in other diseases the fall of temperature appears to be part of the process.

The chart (Fig. 5) represents the effect of a local process in the largest gland of the body upon the general temperature. It is possibly a septic temperature, although the observation was made before the days of bacteriological research. The extreme low temperature is remarkable.

The Diagnostic Significance of Fever. Its Clinical Causes. The presence of fever is of diagnostic importance. It excludes hysteria at once, usually, and generally the feigning of disease. It indicates that one of several morbid processes is present. The morbid processes which give rise to fever are: *First*, infectious diseases, acute and chronic. *Second*, inflammations, which may be confined to the mucous membranes, or to the surface of the skin, or involve the various viscera, or the membranes in relation with the viscera. The fever under these circumstances may be due to irritation of the heat centres or the thermotaxic mechanism by ptomaïnes, or chemical principles derived from the inflamed parts. The inflammation, on the other hand, may be *suppurative* or *septic*. The fever is then higher than in the former condition, and is most marked when pus is closely confined. On account of the local septic process, toxins, or a chemical poison of some kind, are absorbed. The purulent inflammation may be seated in the connective tissue or bones, the brain, the liver or kidney, or the serous membranes. When the local inflammation sets up intense infection of the system by emboli the formation of metastatic abscesses takes place. The fever that attends the process becomes of a peculiar intermittent character, and is known as pyæmic. *Third*, in certain intoxications of the system, as from ptomaïnes in gastro-intestinal disorder, or affections of the liver, and in poisoning from various causes, a fever may be set up. The same mechanism attends the process. *Fourth*, fever may be of central origin, from disease of the brain involving the centres controlling heat, or from disease in proximity to the heat centres. In cases of brain tumor, in cases of apoplexy, and of thrombosis, fever may arise. The centres may also be irritated by direct exposure to external heat alone, or possibly by poisons generated within the system on account of the heat, as in sunstroke. *Fifth*, an irregular form of fever is seen in anæmia and in starvation; while such form is of clinical significance, pathologically it seems to be of the same cause as others mentioned. *Sixth*, a pronounced peripheral irritation or sensation of pain, reflexly altering the thermotoxic mechanism, will produce fever. Hence, in iritis or orchitis a fever

arises out of all proportion to the local inflammation. *Finally*, cases of continued fever exist that have not thus far been classified. One of the nurses of the Presbyterian Hospital with a continued temperature from 100° to 103° was under my care for two months. No general or local condition could account for it. The patient was emaciated. She had had two years of very hard work. Although fever kept up, the appetite was good. Careful feeding of an abundance of food, with rest for many weeks, caused the temperature to fall to normal with complete recovery. I looked upon it as a nervous fever; an expression of exhaustion. Fagge refers to such case. (See article on Fever.)

The Significance of the Initial Stage. 1. In the initial stage of fever sudden rise of temperature to a high degree from a condition of apparent health is against any of the infectious diseases, except scarlet fever. It is of more frequent occurrence in acute gastric or gastro-intestinal catarrh in children than in any other condition in the same class of patients. It may be due to a pneumonia, and is particularly significant if a pronounced rigor attends the rise in adults. In children convulsions may replace the chill. The sudden rise may be due to malaria, in which case it is also accompanied by a chill and followed by free sweating. It may also be due to affections of the throat, to follicular or phlegmonous inflammation of the tonsils. The throat must always be examined in cases of sudden high temperature.

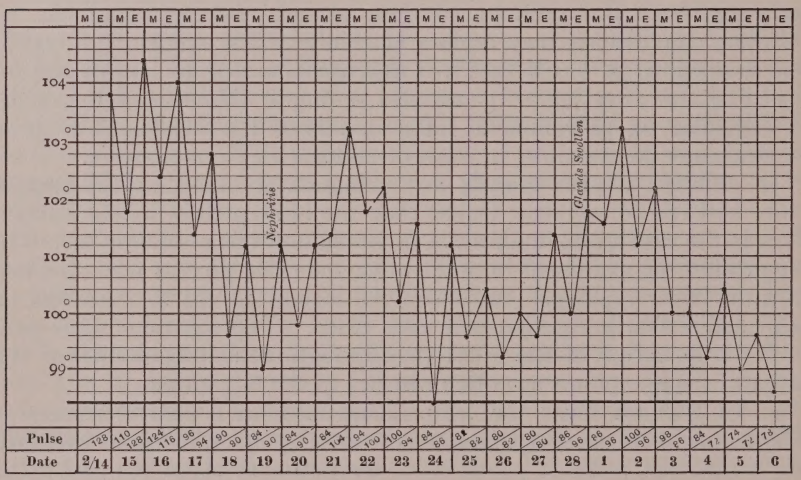
In children if pain attends any inflammatory affection, the temperature will rise to a greater height than the local process would warrant. This is the case with suppurative inflammation of the middle ear. This organ must be examined in order to exclude the process when the temperature rises rapidly. In osteomyelitis and in mastoid abscess the same active febrile reaction will take place. The associate signs point to the true nature of the affection, although it must be confessed that in both, the symptoms are often obscure in the beginning.

2. In typhoid fever the temperature rises in a characteristic way. The temperature ascends by successive evening rises, followed by morning remissions, until it reaches the maximum at about the end of the first week.

The Significance of the Fastigium. In typhoid fever the course of the fastigium is of characteristic significance. From the end of the first, throughout the second week, and sometimes longer, the fever is of the continued type. Subsequently during the third week or later, morning remissions set in, the temperature for a time still rising to the former height in the evening. Then the morning remissions become more decided, the temperature not rising as high in the evening, and so gradually the temperature sinks to and below normal. This course of the temperature in typhoid fever is very far from being invariable; it is modified by indiscretions on the part of the patient or his attendants, and by the necessities of antipyretic or other treatment; nevertheless, the gradual onset of the fever and its long duration are sufficiently common to make them of great value in diagnosis, as, with the exception of tuberculosis, there is hardly any other disease in which a continued fever exists for two or three weeks apart from local inflammation or suppuration.

THE SIGNIFICANCE OF DEFERVESCENCE. A continuance of the fever, the persistence of the fastigium beyond the usual period, is usually significant of the occurrence of a complication. In measles the complication is usually pneumonia. This may take place after the disease has developed, and on account of it the temperature may rise higher than usual. In scarlatina it may indicate acute nephritis, or inflammation of any of the serous membranes, particularly the pericardium or endocardium. Persistence of the fastigium of typhoid fever after the period at which it should decline, if the patient is well nursed and properly fed, usually indicates the occurrence of an inflammatory complication or the development of tuberculosis. In the latter condition the fever is more likely to develop during the afebrile period, the convalescence. Of the inflammatory complications, phlebitis and glandular inflammations are likely to cause persistence of fever after the normal period.

FIG. 6.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104°
103°
102°
101°
100°
99°
Nephritis
Glands Swollen
Pulse
Date
2/14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 1 2 3 4 5 6

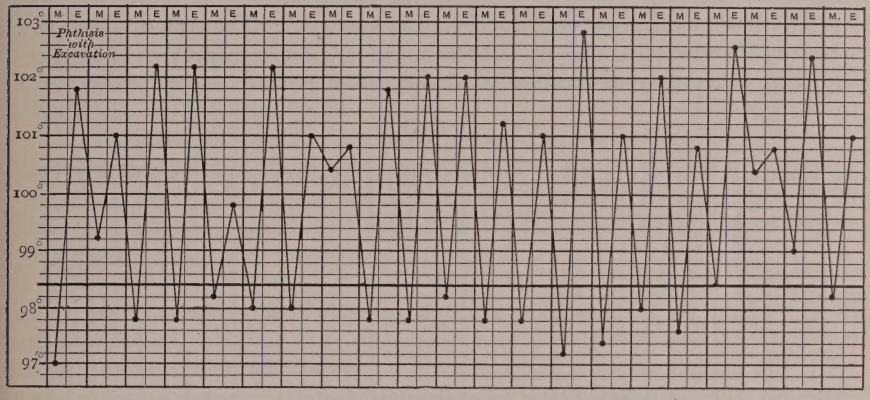
Scarlet fever. Modification of temperature by complications. Nephritis on the ninth day.

THE SIGNIFICANCE OF A SUDDEN FALL OR OF SUBNORMAL TEMPERATURE. The occurrence of the normal or subnormal temperature in a person who has previously had high fever signifies the occurrence of crisis if the time for that event has arrived, as in pneumonia; or of a grave complication, causing shock to the system. In typhoid fever this unusual drop in the temperature will take place if there has been a hemorrhage from the bowels, or perforation, or the occurrence of peritonitis. It must not be confounded with the sudden falls of temperature that occur in the typhoid fever of children, corresponding to the onset of convalescence. These occur earlier in the period of the disease than with adults.

THE DIAGNOSTIC SIGNIFICANCE OF THE TYPE OF THE FEVER. *Intermittent Fever.* The temperature range has been observed for a number of days and an intermittent type of fever ascertained to be present. The representative of the type is seen in *malaria*, but it is simulated by a number of conditions: (1) In certain cases of *typhoid*

fever and of *relapsing* fever the type is intermitting or paroxysmal. The same type of fever is seen (2) in *suppuration*, particularly if the pus is confined; (3) in *ulcerative endocarditis;* (4) in tuberculosis. *a.* This may occur in tuberculosis in the earlier stages. The primary seat

Fig. 7.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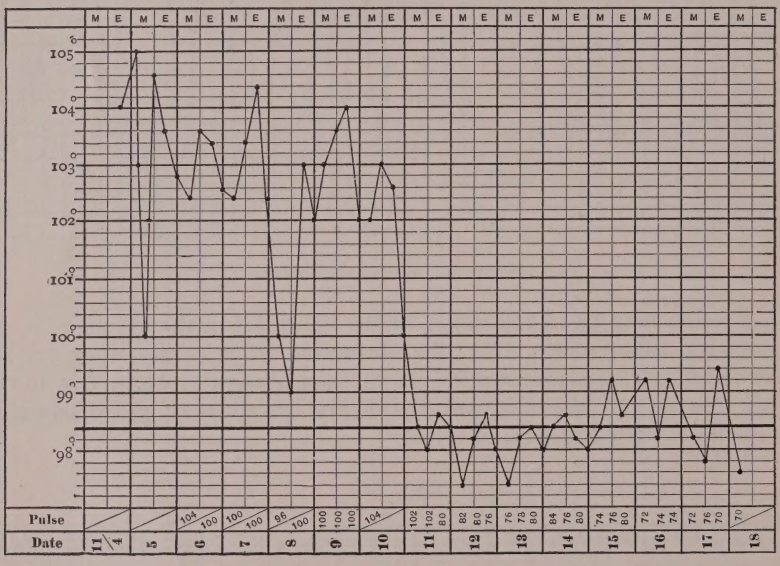
catheter or sound, but it may also occur when there is suppuration in the genito-urinary tract. (8) *Hepatic intermitting* fever is a form of frequent occurrence and of great diagnostic importance. It may be due to the presence (*a*) of gall-stones somewhere in the biliary ducts, usually with obstruction; (*b*) to the presence of suppuration in the canal with or without obstruction; (*c*) to the obstruction of the biliary passages by external pressure without the occurrence of suppuration; (*d*) inflammatory affections of the liver, as abscess, and forms of cirrhosis. Rarely it occurs in rapidly growing cancer. (See Fig. 5.) (9) Intermitting fever may also occur from the prolonged use of *morphia*.

FIG. 9.



Pneumonia. Sudden rise. Termination by crisis. Pseudo-crisis also seen.

Of the above-mentioned varieties of paroxysmal or intermitting fever, those of most common occurrence are due to suppuration, pyæmia, to ulcerative endocarditis, to tuberculosis, and to hepatic disorder. In this class of cases, in addition to the paroxysmal rise in temperature, rigors and sweating frequently precede and follow the paroxysm, as in cases of intermittent fever. The diagnosis from true intermittent fever can be established at once by an examination of the blood, which reveals in the latter the plasmodia of Laveran.

Remittent fever. Fever of a remittent type occurs in many of the conditions in which intermittent fever is present. It is characteristic of one of the forms of malaria. It is most frequently encountered in tuberculosis of the lungs. The remissions usually occur in the mornings, but the order may be reversed. The same type of fever is met with in puerperal fever, pyæmia, and septicæmia, and in local suppurations, such as abscess of the liver and empyema. A continued fever may be made to resemble a remittent by antipyretic treatment, on

account of which abnormal drops in the temperature take place. Remissions characterize the decline of the continued fevers, particularly typhoid, during the period of lysis.

Continued fever. Continued fever is met with in lobar pneumonia, typhoid fever, typhus fever, erysipelas, and tuberculosis of the lungs at times. In acute lobar pneumonia the temperature rises rapidly, and in a few hours from the initial chill reaches 103° or 105°. The morning and evening temperature varies but little, usually not more than one or two degrees until a crisis occurs, in from four to eight days. The temperature then falls to or slightly below normal, and does not again rise. (See Fig. 9.)

A marked remission in the fever sometimes occurs on the fourth day before the actual crisis; the temperature falls to 100°, and again rises to 103° or 104°; remains at that level for twenty-four or forty-eight hours, when the true crisis occurs. The fall of temperature or defervescence (crisis) may occur in a few hours.

Diagnostic Significance depending upon Age and Sex. The significance of a high febrile change in children is not so great as in adults. That is, the high temperature is not so important, inasmuch as children are liable to have sudden rises of temperature to a great height; and a higher temperature may persist for some time without the effects upon the tissues which occur in adults. In women of nervous temperament the temperature is also likely to rise to a great height without marked cause or serious result.

The General Musculature. The state of the muscles must always be learned. In the discussion on emaciation it was referred to. A few words more seem necessary. It must be remembered that a person can be obese and yet have poor muscular development, or have little fat and fair muscle. General lack of muscular development, or muscular weakness, is a most important sign of malnutrition, and when recognized will explain the nature of many symptoms. Weakness of the muscles of the spine, with resulting curvature, or inability to keep in the erect posture, is sufficient cause for the occurrence of neuralgic pains in the course of related nerve trunks, and for the displacement of organs within the thorax or abdomen, on account of which functional disturbance has arisen. Various uterine displacements and functional disorders may be mitigated by toning up the nutrition of the muscles of the trunk. Forms of indigestion, sluggishness of secretions, particularly of the bowels, follow in the wake of debilitated muscle and pass away as such muscles gain tone. It may be that the indigestion has not taken place because the muscles are weak, although in a measure there is relation between them; but the weak, flabby muscles are pronounced indications of a state of the system which certainly does cause the development of such conditions. The observation of muscular deficiency leads to correct lines of treatment. Atrophy occurs because of disuse, because of sedentary occupation, or a life of ease and luxury with improper nutrition. It is sure to follow improper assimilation in its most prominent form, as seen in *anorexia nervosa*.

General Abnormal Vital Conditions. Fits, or Seizures; Collapse; Coma. General observation of the exterior at once reveals to us the occurrence of fits, should they have taken place, or of alterations of the consciousness of the individual. The two often go hand-in-hand, but in some instances, as in fainting fits, consciousness is not lost. The following indicate the forms of each that may be met with. Only those are mentioned which occur instantaneously, and for which the doctor is called. For their symptomatology and diagnosis the appropriate sections on special diagnosis must be consulted.

I. *Fits with unconsciousness:*

1. *Syncope.* The face is pale but calm, the pulse feeble or imperceptible, the extremities cool, nausea or hurried breathing may precede. The breathing is quiet in the attack. The pupils respond to light. No pain. (See Heart Disease.)

2. *Apoplexy.* (Spasm is sometimes associated.) Head pain, congested face, hemiplegia, facial palsies, pupils irregular and irresponsive, cornea not sensitive, incontinence of urine; pulse strong, full; arteries hard.

3. *Epilepsy.* (1) "Haut mal:" aura, convulsions; (*a*) tonic, respiratory muscles affected, face livid, stupor afterward; (*b*) clonic, tongue bitten, stupor follows. (2) "Petit mal:" pallor sudden, no convulsions.

4. *Infantile convulsions.* Usually reflex from indigestion; may be the onset of a specific fever, or due to high temperature.

5. *Puerperal convulsions.* Headache, amaurosis, œdema, suppressed urine, albuminous; clonic convulsions, tongue bitten, complete coma. (See Uræmia.)

6. *Uræmia.* Unilateral or bilateral clonic convulsions. (See Renal Disease.)

7. *Alcoholism, opium, and sunstroke.*

8. *Organic brain diseases* (syphilis, tumor, softening, etc).

II. *Fits with partial or no loss of consciousness.* Faintness, angina pectoris, hystero-epilepsy, focal or Jacksonian epilepsy, hysteria, cerebral embolism, thrombosis or hemorrhage, spasms of various kinds.

III. *Fits with vertiginous movement.* The forms of vertigo are gastric, aural and labyrinthine (Ménière's, also paroxysmal), ocular, cerebellar, congestive of the brain (reflex), epileptic.

IV. *Collapse.* This is likewise a condition that may be present with the immediate onset of the disease and be noted by the observer on the occasion of his first visit.

The symptoms are those of prostration, with partial loss of consciousness, or the mind is perfectly clear. The face is pale, pinched, and bathed with perspiration (see Hippocratic Facies). The skin is cool and clammy. The hands are cold. The skin is wrinkled. The eyes are sunken and encircled by dark rings. The voice is weak or suppressed. The pulse is rapid and thready, or may be absent at the wrists. The heart sounds are indistinct. The temperature falls. The respiration may be hurried, or shallow, sighing and gasping. The urine is scanty or may be absent. Collapse is due to hemorrhage, external or internal; to perforation of abdominal viscera, to peritonitis, to excessive watery discharge, as in cholera or serous purging. It may be due to pernicious malarial fever. Coma attends this form.

V. *Shock* is a condition in which the vital powers are blunted or stunned, with or without mental terror or anxiety. In injury, surgical operation, hemorrhage, severe pain, undue mental and emotional strain, it is likely to be seen. Its presence points to a grave antecedent condition, near or remote. The symptoms are those of collapse.

B. Local Examination of the Exterior.

The appearance of the face and its expression are observed. The shape and size of the head and the mode of carriage are noted. Abnormalities must be taken note of, and in addition, local areas about the face, as the eye, nose, and ear, particularly inspected. Passing downward from the head, the neck should be examined, and from thence a local examination of the extremities, independently of the skin, made. Following this, the bones and joints and the muscles should be studied in regular order.

The Face and the Facial Expression. (See Nose and Mouth in respective chapters on special diagnosis.) The face is a mirror in which is reflected all degrees of ill health, from that which amounts only to temporary indisposition and depression up to the gravest cachexia. The face reflects also the degree of intelligence of the patient and his mental condition at the time, as well as his emotions, and in a large measure his character. The face is usually a pretty good index of the temper of the individual; benevolence, amiability, and purity are written as plainly on some faces as anger, lust, dishonesty on others.

All varieties of mental aberration are reflected in the face; the suspicious, at times revengeful, look of the delusional monomaniac; the wild look and excited manner of the maniac; the plaintive, depressed, injured look of melancholia; the vacant, listless, peaceable, animal-like look of dementia—a look which changes for animation only at the sight of food or some coveted luxury. All these expressions come to be recognized very readily by those who see much of the insane.

The face frequently affords us valuable information concerning the health, habits, and temperament of the individual. Everyone is familiar with the bright eye and animated countenance of a friend which lead us to say, "You are looking very well to-day," and with that slight pallor, diminished clearness of the conjunctiva, with perhaps a dark circle under each eye, which leads us to infer that he is depressed or has passed a sleepless night. The face also gives unmistakable evidences of alcohol by its bloated appearance, injected or glassy eye, dull expression, and nervous manner when addressed suddenly.

Full-blooded persons, disposed to endarterial changes, frequently as the result of gout, often have, at a little distance, the *ruddy* appearance of blooming health. Closer inspection, however, shows that the ruddy color is due to a dilated or congested condition of the minute blood-vessels. This condition, when associated with high tension in the arteries and accentuation of the aortic second sound, is highly suggestive of chronic nephritis. (For color, see Skin.)

Moreover, the face tells of the presence or absence of pain, and, to a

certain extent, of its character. Everyone has witnessed the sudden contraction of the brow and eyelids, and the involuntary sucking in of the breath when someone has bitten upon a tender tooth. Other faces bear the imprint of long-continued, more or less constant suffering. According to Eustace Smith, pain in the head in children is indicated by contraction of the brows; pain in the chest, by sharpness of the nostrils; and in the belly, by a drawing of the upper lip.

It will be seen that the expression, the color, and the outline of the face are valuable indications of disease.

The master mind in clinical medicine, the late Austin Flint, Sr., tersely described the various appearances of the face in disease, with their clinical significance, as follows:

The Facies of Renal Disease. In some cases of acute albuminuria, and of chronic parenchymatous nephritis—the large white kidney of Bright—puffiness of the face from œdema, with notable pallor, renders the aspectic highly diagnostic.

The Malarial Facies. Pallor of the face, sallowness, and slight puffiness, if renal disease be excluded, point to malarial disease.

The Facies of Carcinoma. Notable anæmia, a waxy or straw-colored complexion, and more or less emaciation, in combination, render the aspect marked in some cases of malignant disease. In a patient over forty years of age this aspect has considerable diagnostic import, although it is by no means always present when malignant disease exists.

The Typhoid Facies. In the middle and later periods of typhoid fever the countenance is often dull, besotted, expressionless. This facies may be present in the typhoid state, which is incident to diseases other than typhoid fever, *e. g.*, pneumonia. Coexisting with a dusky hue of the skin and congestive redness of the conjunctiva, it distinguishes typhus, as contrasted with typhoid fever.

The Facies of Acute Peritonitis. The upper lip raised so as to expose the front teeth, gives an aspect which characterizes, in a certain proportion of cases, acute peritonitis. It is often wanting, but when present it is strongly diagnostic.

The Facies of Acute Pneumonia and Hectic Fever. Circumscribed redness of one or both of the cheeks, with abruptly defined borders, is diagnostic of acute pneumonia. If it be observed in a case of chronic pulmonary disease, it denotes the so-called hectic fever, and is a sign of phthisis.

The Facies of Exophthalmic Goitre. Projection of the eyeballs, giving to the face a remarkably staring and sometimes ferocious expression, conjoined with enlargement of the thyroid body and frequency of the pulse, is distinctive of the affection known as exophthalmic goitre—Graves' disease and Basedow's disease.

The Choleraic Facies. In the collapsed stage of cholera the face is contracted, sometimes wrinkled; the cheeks are hollow, the eyes sunken, the skin is livid, and the expression denotes indifference. This combination of traits is quite distinctive. They are, however, to a certain extent combined in the state of collapse which occurs in some cases of pernicious intermittent fever, and in other pathological connections.

The Hippocratic Facies. This facies denotes the moribund state. The skin is pale, with a leaden or livid hue; the eyes are sunken, the eyelids separated, and the cornea loses its transparency; the nose is pinched, and the eyes are contracted; the temples are hollow, and the lower jaw drops. Hippocrates described this facies in graphic terms, and the name Hippocratic has ever since been used to designate it.

The Face in Children. Inspection is even more important in the case of children than in adults. The pale, pinched, weazened face of some babies who have snuffles, ulcers at the corners of the mouth, and look prematurely aged, is characteristic of *inherited syphilis.* In *rickets* the head is unusually large with flattened vertex, projecting *forehead,* and open fontanelle. In *hydrocephalus* the head becomes very much enlarged, the eyes prominent, the bones of the *face* remaining small, the expression vacant (see p. 122). In *measles* the red, swollen face, the reddened, weeping eyes, and running nose make a very striking picture. An irritating, excoriating discharge from the nose in a child may indicate the existence of a nasal *diphtheria.*

The Face in Nervous Disease. The face also often tells of the existence of some organic nervous disorders.

In *peripheral facial palsy* the paralyzed side of the face has a staring, vacant expression, owing to the fact that the eyelids are motionless. The angle of the mouth on the affected side is depressed. The whole paralyzed side is devoid of wrinkles, has a smoothed-out, glazed appearance; tears flow over the cheeks, and saliva dribbles from the corner of the mouth. The contrast with the normal side is most marked when the patient smiles or frowns.

In *glosso-labial palsy* there is progressive palsy, with tremulousness of tongue and lips; progessive failure of articulation, and dribbling of saliva. Sometimes the patient is able to open the lips, but unable to close them without the aid of the hand.

A slow, hesitating, thick manner of speaking, with a tendency to slur the labial and lingual consonants, when associated with irregularity of the pupils, slight tremulousness of the lips, and the loss of the fine adjustment of other muscular movements, such as writing, is very suggestive of *general paralysis of the insane,* especially when the condition develops in a middle-aged man.

Facial hemiatrophy is a peculiar affection, characterized by progressive wasting of the bones and soft tissues of one side of the face. The disease is rare; it begins, as a rule, in childhood, and may develop in later life. The local change is diffuse, although, in some instances, it starts at one spot in the skin and spreads, involving, in succession, the tissues underneath. The skin changes in color, and the hair falls. The eye is sunken on the affected side on account of wasting of the tissues of the orbit. Of the bones that waste, the bone of the upper jaw atrophies to a more advanced degree than the others. On account of the wasting of the alveolar processes, the teeth become loose and fall out. The wasting is sharply limited to the middle line. The disorder is easily recognized. The patient looks as if the face was made up of two halves from different persons. It must not be mistaken for the facial asym-

metry that is associated with congenital wry-neck. The contraction of the sterno-mastoid muscle from birth distinguishes the affection.

(For spasm and contraction of the muscles of the face, see Disease of Cranial Nerves.)

Having noted the expression of the face and observed the general and local color, the outline of the face, with any change in the shape of the head, should be observed. The changes in both, as seen in rickets, have been described. The striking changes in acromegalia, myxœdema and osteitis deformans have also been described in sections referring to these affections.

Enlargement of the Face. *Swelling.* Other changes in the outline of the face and skull are significant. The face is swollen and deformed in erysipelas and smallpox. The specific eruption serves to distinguish each one. The puffiness of the eyelids and general swelling of the face, which arises in the course of Bright's disease, will be referred to. (See Œdema.)

In *mumps* the swelling is characteristic. It usually begins on one side first. The swelling of the parotid gland is observed in front of the ear, then it extends below and around it and behind the ramus of the jaw. Unless there is much collateral œdema, the outline of the gland is preserved. The lymphatics may or may not be swollen. It is tender and boggy, not indurated. Viewing the face from the front, the mid-lateral aspects are seen to bulge. The ears stand out from the head.

Œdema of the face occurs in *trichinosis.* It occurs at two periods in the course of the disease. It is seen in the eyelids in the beginning of the disease, and disappears after a few days. Later in the disease it returns, with pain, tension, and restriction of the movement of the eye-muscles. The œdema may be due to infection of the muscles by the parasite or may be of vasomotor origin.

Hair. The hair often indicates the state of the nutrition of the individual. Changes in it may be significant of syphilis or other internal morbid processes. The abnormal growths and changes in the texture due to local parasitic disease will not be referred to. Undue and rapid falling out of the hair in patches, known as alopecia, is indicative of syphilis and of profound intoxication by the virus of this disease. The hair can be pulled out in large masses without difficulty or pain. This falling of the hair must not be confounded with the excessive falling out which takes place in the convalescence of acute disease, and particularly of typhoid fever, nor with that following an attack of gout or erysipelas.

Color of the Hair. Obscure paralysis or anæmia may be explained by noting the artificial coloring of the hair. Repeatedly lead and other poisonings have arisen from the use of hair dyes. Other changes in the color are not specially significant, although early gray hair may go hand-in-hand with premature endarteritis. The term "canities" is applied to the diminished development of pigment. Premature gray color in defined patches occurs in nerve lesions, as paralysis of one of the branches of the fifth pair, and is a trophic change.

Local Affections of the Skin of the Face. Comedones are papular elevations with a central pit of dark color, due to accumulated dirt. Milium, from obliteration of the ducts, consists of small, rounded papules of whitish color. They are familiar, but are not of special significance. In molluscum the entire sebaceous gland is distended by altered secretion. Acne is a papular affection, inflammatory in character, appearing at the seat of comedones, and may be indurated or pustular. In the latter instance pits are left behind. It is commonly seen in gouty subjects, in chronic dyspepsia, or liver disturbance.

The *sebaceous glands* of the skin of the face merit but a passing notice. Deficiencies or excesses of secretion, or alteration of it, are usually due to local causes. Excessive secretion of sebaceous matter, known as seborrhœa, or steatorrhœa, is seen in two forms. First, with oily exudation; second, with drying of the secretion and the formation of crusts. It may be more pronounced in strumous subjects. The opposite condition, or asteatodes, is seen in wasting diseases, particularly diabetes, and in xeroderma and ichthyosis.

The Cranium. The change in shape that takes place in general bone affections has been referred to (see preceding pages for rickets, acromegalia, osteitis deformans). The peculiar shape due to deformities of congenital origin, or deficiency of the bone plates, are not within the province of this work. By palpation the fontanelles are examined, the presence of bosses detected, and the loose plates adjacent to the sutures ascertained. The term *craniotabes* is applied usually to the occurrence of this condition in early rickets.

Fontanelles. *Prominence* or *fulness* is seen in hydrocephalus and other brain affections in which there is increase of internal pressure. *Depression* of the fontanelles occurs in general atrophy, marasmus, and in wasting diseases generally. It is present in collapse, and is of prognostic omen. The fontanelles are not changed in rickets, a point of distinction between this affection and hydrocephalus and enlargement from other internal causes. The *bones* of the cranium may be thickened; they may be the seat of periostitis, of necrosis, and caries. Necrosis and caries of the frontal bone is almost pathognomonic of syphilis. Necrosis of the jaw bone belongs to phosphorus poisoning. The mastoid and petrous portions of the temporal bone should be examined in many affections. The symptoms that should call our attention to these bones are pain and tenderness over the mastoid, rigors and fever, with thrombosis of the cerebral sinuses, characterized by pain in the head, convulsions, and strabismus. Examination in the region should extend to the occipito-atlantal articulation. Disease of this articulation, and particularly tubercular disease, causes stiffness of the neck, or falling forward of the head. On account of the stiffness, associated difficulty of deglutition and pain, the writer has seen it mistaken for retro-pharyngeal abscess.

The expression and contour of the face is of much significance in cerebral disorders.

Affections which cause an increase in intra-cranial pressure cause, also, striking external features, as in hydrocephalus.

Hydrocephalus. The external enlargement of the skull is very conspicuous, and the undue proportion of the cranium to the face is striking. The cranium is rounded or globular in shape, and the fontanelles are seen to be very large, tense, and bulging, and the sutures widely separated. The disproportion in size of the face and head is increased by the projection of the front portion of the skull. The axis of the eyes is directed downward, and they are partly covered by the eyelids, because of the oblique direction of the orbital plates. The head is supported with difficulty; the eyeballs roll from side to side. There is frequently strabismus. The skin is stretched tightly over the cranium, and the hair is scanty. (See Fig. 10.)

FIG. 10.

Congenital hydrocephalus. Female, aged seventeen. (The thinness of the hair could not be represented.)

The enlargement of the head must not be confounded with rickets (see under Skeleton) or enlargement and thickening of the bones. In the former the head is square in shape, not globular, and the fontanelles, though large, do not bulge. Other signs of rickets aid in the distinction. Gowers states that thickening of the cranial bones may simulate hydrocephalus at almost any age. He thinks it doubtful whether the nature of the latter rare cases can be ascertained during life. The thickening that attends *osteitis deformans* and *acromegalia* have been already described.

The Lips. The *color* of the lips is pale in anæmia, and livid in cyanosis from chronic lung or heart disease with feeble circulation. Vesicles (herpes) are apt to appear upon them in common colds, in certain febrile diseases, particularly pneumonia, and with many women during or immediately following menstruation. A child with hereditary syphilis may show ugly fissures, or the scars which result from them, at the angles of the mouth. In facial palsy the angle of the mouth on the paralyzed side is depressed and free from wrinkles. In glosso-labial laryngeal palsy the lips tremble, twitch, and may have to be closed with the fingers after they have been opened. In general paralysis of the insane the lips tremble, and speech is "thick," hesitating, and uncertain, with a tendency to elide syllables and slur the labial consonants.

The Eye. Appearance of the eyelids in œdema has been described. The dropsy may accumulate during the night in little bags under the lower eyelid. It is seen in the morning on rising, and disappears by night. (See Œdema.) It must not be confused with the morning puffiness that seems to be natural to some individuals, or the swollen face that succeeds a debauch. We sometimes see a peculiar change of the skin of the eyelid due to xanthelasma. In addition to its occurrence in this situation, the palms of the hands, the flexures of the fingers, and the inside of the mouth are affected (see under Tongue). On the eyelids are seen patches slightly elevated, of a yellowish color, irregular in shape. They are slightly sensitive to the touch but not indurated. The cuticle is healthy. They are due to oil deposited in the neighborhood of the hair follicles, in the substance of the cutis. Sometimes they are arranged in the form of tubercles as large as a pea.

Drooping of the eyelids may occur from paralysis of the third nerve. It is known as *ptosis*. (For this and affections of the oculo-motor and optic nerve, see under The Eye—Nervous Diseases.)

The Open Eye. This is known as *lagophthalmos*. It is due to paralysis of the orbicularis palpebrarum. It is present in more or less degree in exophthalmic goitre.

Exophthalmos. The eyeball protrudes more or less from the socket in tumors of the nose or orbit.

In exophthalmic goitre both eyeballs protrude, and with the change in the appearance of the neck give to the patient the so-called ferocious appearance. The protrusion of the eyeballs is readily recognized because it is bilateral. The so-called Von Graefe's sign further aids in the diagnosis. This sign consists in lagging of the upper lid in movements of the eyeball. When the patient looks down the lid does not readily follow the movements of the ball downward.

Stellwag's sign is the third sign of significance in exophthalmic goitre. There is undue exposure of the cornea. One or both signs may be absent in cases of exophthalmic goitre.

Sunken Eyes. Sunken eyes are due to atrophy of the fat of the socket in phthisis or wasting diseases. It is most pronounced in the sudden atrophy that occurs in cholera from loss of water. It is also seen in peritonitis and collapse from other causes.

Examination of the Conjunctivæ. The conjunctivæ may be

the seat of inflammation from local causes. Its occurrence in the course of general or internal disease concerns us. It is often seen in disease of the brain or the meninges, and sometimes it occurs early in the course of the affection. In tuberculous meningitis purulent conjunctivitis is of common occurrence. Usually one side is more highly inflamed than the other. Along with other symptoms of involvement of the cranial nerves, the conjunctivitis is of diagnostic significance. In measles conjunctivitis is seen early. In typhus fever it is a constant sign and serves to distinguish the affection from typhoid. In yellow fever the mild conjunctivitis causes the watery ferret-eye. The conjunctiva is used to determine the degree of sensitiveness of a patient who is more or less comatose.

The Color. The normal color of the ocular conjunctiva is clear white. In jaundice it is yellow. It is yellow in small areas from fat in the obese and aged. The fat is in cone-shaped areas. The pearly sclerotic of chlorosis, the dead-white color of anæmia, as in Bright's disease and phthisis, are striking in these affections. The palpebral and outer conjunctiva is the seat of hemorrhage in epilepsy, whooping-cough, asthma, and of hemorrhagic infarcts in ulcerative endocarditis. (See Disease of Cranial Nerves for movements of eyeball, the iris, appearance of the retina, etc.)

The Cornea. Ulceration of the cornea in addition to other causes occurs as a trophic lesion due to paralysis of the first branch of the trifacial nerve. It may occur in paralysis of the eyelid from exposure. Opacities result from such ulceration or may be due to syphilis. In congenital syphilis the remains of keratitis are frequently seen. The "arcus senilis" is observed in the circumference of the cornea at its junction with the sclerotic. It is a distinct arc but is not always a complete circle. The cornea is hazy and may have fat granules. The eyelids must often be lifted to recognize it. Its edges are ill-defined. In contradistinction to this, Fothergill calls attention to the false "arcus senilis"—a well-defined ring which encircles the pupil; but the cornea is always clear and the person in good health, although aged. The true arcus senilis is seen in the gouty, in arterial sclerosis, and in nephritis. It is an early indication of degeneration of the arteries.

The Ear. In an exhaustive general examination, either with the object of determining the body health for life insurance, or in order to determine the cause of ill health, the external ear should always be examined. This should be particularly the case in inflammations of the meninges or other disease of the brain. In otherwise unexplainable cases of pyæmia or of pyæmic symptoms (alternating chills and fever), the presence of discharge from the ear should be inquired for, as very frequently middle-ear disease results in inflammation of the mastoid, and from thence the sinuses and membranes of the brain adjacent become inflamed, or the suppuration may be the primary focus from which general infection takes place. It may not be possible in all cases to observe a discharge. It may have diminished or disappeared on account of the fever. Tenderness and œdema over the mastoid, and direct inspection of the ear drum, by which a perforation, or other charac-

teristic changes may be seen, point to the occurrence of suppuration in this situation. It must not be forgotten that in fractures of the skull a bloody discharge from the ear may take place. In cases of coma from injury, or if of obscure origin, the ears must also be examined.

The External Ear. From the exterior of the ear we derive but little data of diagnostic significance. It is true the thin ear may show the anæmic or chlorotic hue more strikingly than other portions of the body; or the opposite condition may be more vividly shown. *Hæmatoma auris* is seen in general paralysis of the insane and other forms of insanity. It is a tropho-neurosis. The ear is thickened and deformed on account of effusion of blood between the cartilages and the perichondrium. It is discolored and simulates the subcutaneous effusion due to injury. Apart from color changes *tophi* are observed on the external ears of patients with a gouty diathesis. They are small, hard, gritty accretions seen on the external ears along the margin, or in the depressions. They consist of urate of soda.

The function of *hearing* must be inquired into and its acuteness tested. This may be done with the voice, with the watch, and with the tuning-fork. The voice may be heard well in some cases when the ticking of a watch can be perceived with great difficulty. The tuning-fork is used to determine whether the deafness is due to (1) obstruction or (2) disease of the internal ear. If it is due to obstruction the fork is heard better on contact with the skull than when the sound is heard in the natural way through the ear. Deafness of this character is always due to disease of the external meatus, the tympanic membrane and middle ear, or the Eustachian tube.

Deafness due to disease of the internal ear may be due to affections of the labyrinth, as caries and necrosis, or diseases of the auditory nerve. The tuning-fork is not heard on contact with the skull. The auditory nerve may be diseased in its course, or the auditory centre may be affected. Tumors, meningitis, hemorrhage, and infectious diseases may involve the auditory nerve, while the auditory centre is affected by tumor, meningitis, abscess, or hemorrhage. (See under Cerebral Localization.) It must not be forgotten that certain drugs, as quinine and the salicylates, may cause deafness.

The Neck. *Shape and size of the structures.* The position of the trachea and larynx, the seat and size of the thyroid gland, and the appearance of the vessels of the neck, should be observed. The trachea and larynx occupy the median line in health, but may be deflected to the right or left. The deflection is more readily noticed at the lower part of the neck, and can be ascertained by fixing the relationship to the adjacent muscles. The change in position is due to disease within the thorax. An aneurism or mediastinal tumor may cause this alteration. In cases of chronic fibroid phthisis the trachea is pulled to the side of the affected lung. *Movements* of the larynx and trachea are observed, and when in excess and associated with dyspnœa the source of the dyspnœa is in the larynx. When, on the other hand, they are not moved, or indeed remain fixed notwithstanding violent efforts at

respiration, the dyspnœa is due to disease in the mediastinum. This form of dyspnœa occurs from enlargement of the mediastinal glands or from aneurism pressing upon a bronchus. Observation of the condition of the trachea and larynx is made by inspection and by palpation. Both are employed in diseases of the larynx (see Larynx) and the latter in order to detect the physical sign due to tugging or drawing on the trachea by disease within the thorax. Tracheal tugging may be seen, but is usually determined by palpation. It is particularly characteristic of aneurism of the descending portion of the aorta. The aneurismal sac presses upon the bronchus, and on account of its relationship with each pulsation of the vessel, the tugging or pulling downward of the trachea can be felt. (See Diseases of the Vessels.)

Thyroid Gland. It may be enlarged or diminished in size. The atrophy of the gland is shown by absence of fulness, which should otherwise be present in the neck of the individuals of the age of the patient under examination. (See Myxœdema and Acromegalia.)

Enlargement of the Thyroid can be detected without much difficulty. It may be limited to one lobe, or both lobes may be affected. It may vary in size from a small localized swelling to large masses which fill the median and lateral sides of the neck, pressing upon the trachea. On palpation the swelling may be soft or hard. In the fibrous forms the swelling is not very large and is very much indurated. In the cystic forms of the thyroid enlargement fluctuation may often be detected ; it may be localized to a small area of the lobe or may be detected over the entire affected lobe. On palpation in some cases a purring or thrill is transmitted to the fingers. The thrill is synchronous with the heart's action, due to great vascularity of the parts. Auscultation over the gland when a thrill is present reveals a murmur systolic in time and low in pitch.

Causes. Enlargement of the thyroid gland may be due to simple hypertrophy, to fibro-cystic enlargement, or to enlargement in which the vascularity is more prominent, as in exophthalmic goitre. In simple hypertrophy the enlargement is often intermittent, increasing in size at each menstrual period, or coming on in pregnancy, to disappear after labor. It may then disappear entirely, or again return at the menopause. The fibro-cystic enlargement which occurs in countries in endemic form persists. The enlargement which is chiefly due to dilatation of the blood-vessels is usually seen in exophthalmic goitre, and can easily be recognized by the association of the remarkable signs of this affection. (See Exophthalmic Goitre.)

Enlargement of the thyroid gland must be distinguished from enlargement due to other causes, as cancer, sarcoma, or adenoma. It must also be distinguised from other tumors in this region. It particularly must not be confounded with enlargement on the right side due to an innominate aneurism. (See Aneurism). The distinction can usually be made without difficulty. (For lymphatic glands of neck, see The Glands).

The Vessels of the Neck. The large veins of the neck form an accurate clue to the state of the circulation within the veins. Their close proximity to the heart in the direct line which the blood takes to

reach the heart causes other changes which indicate the state of the circulation in that organ. (For a description of these changes see Arteries and Veins.)

The observation of the *thorax* and *abdomen* will be considered under sections devoted to affections of the respective regions.

The Extremities. The Hands. *Color.* Observations of the color of the *hands* is of service in estimating the general hue and color of the individual, as changes are noted earlier in the distant points of the circulation. (See the Skin—color.) *Shape.* Changes in the shape

FIG. 11.

Pseudo-muscular atrophy. Claw-hand. (GRAY.)

are pronounced in many affections. The spade-like hands of myxœdema have been referred to, the peculiar shape of the hands in acromegalia and pulmonary osteo-arthropathy described, and the appearance in rheumatoid arthritis also discussed. In *progressive muscular atrophy* (chronic anterior myelitis) the shape of the hands is peculiar. The French name *main-en-griffe* is applied to it. Both hands are affected, although it may have begun in one before the other. From wasting of the muscles voluntary power is lost. The thenar muscles and the interossei are the first to suffer. The thenar eminence becomes flattened, the base of the first metacarpal bone more prominent. The atrophy of the abductor indicis is so conspicuous that the normal prominence near the thumb when it is adducted gives place to a hollow beside the metacarpal bone. There are marked depressions between the metacarpal bones and the flexor tendons of the hands. The phalanges assume positions dependent upon the degree of atrophy of the flexors or the extensors of the forearm. The extensors on the ulnar side usually atrophy the most, and the extensors of the phalanges of the thumb more than that of its metacarpal bone. A peculiar claw-hand is produced on account of these contractions.

RHEUMATOID ARTHRITIS. The shape of the hand somewhat resembles that of muscular atrophy. While there is considerable atrophy of

the muscles there is also change in the ends of the bones and joints. The ends of the bones are enlarged and the cartilages undergo atrophy. The joints of the phalanges may be swollen and the tissues infiltrated prior to the destruction of the cartilage. This may have been present for a long time, increasing in amount at different periods with pain and tenderness. The joints gradually become more immobile, the infiltration disappears, and the enlarged ends of the bones become more prominent. More or less ankylosis develops, and on motion crepitus and grating is felt on account of the eroded cartilage. Osteophytes may form in the

FIG. 12.

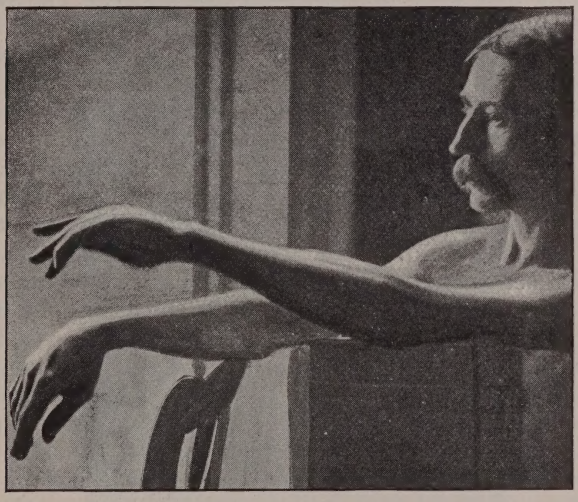
Rheumatoid arthritis. The phalangeal joints are swollen; many are ankylosed. The wrist is stiff. The muscles are atrophied; the forearm muscles much wasted.

tendons, so that the joint becomes more completely locked. Atrophy of muscles supervenes on account of the disease of the joint. Sometimes the wasting is very extreme and gives the hand the appearance that is seen in pseudo-muscular atrophy. The general symptoms that attend each affection serve to distinguish them. Rheumatoid arthritis is easy of recognition when the other joints are involved in the process.

CONTRACTIONS of the hand may often be observed from other causes than the ones just mentioned. Temporary contractures occur in tetany, in temporary hemiplegia or monoplegia, and in paralysis of the extensors. So-called *wrist-drop* is seen in *peripheral neuritis*, particularly in the form due to lead. The hand hangs from the wrist on account of paralysis of the extensor muscles of the forearm. Both hands may drop, although dropping of one is seen from a few days to a few weeks before that of the other. It develops gradually. At first the patient cannot extend the fingers at the metacarpo-phalangeal joints. The thumb also suffers, and the weakness of the extensors is most

marked on the ulnar side. At the beginning, if the first phalanges are passively straightened the distal phalanges can be extended by the unaffected interossei muscles. The loss of power extends to the wrist. The extensors of the wrist do not suffer equally. Those of the radial side are affected first. When the paralysis is complete the hand drops and cannot be brought to the level of the forearm. It may be noted that if the fingers are flexed passively the patient is able to close the fist as long as the special extensors of the wrist retain power. If, however, the fingers are extended the wrist cannot be extended. The muscles affected, therefore, are the common extensor of the fingers, the

FIG. 13.



Photograph of a case of lead-paralysis affecting the extensor muscles. (GRAY.)

extensor indicis, the extensor of the phalanges of the thumb, and those of the wrist. The flexors of the fingers are unaffected. The continued over-flexion of the carpus produces slight displacement backward of the carpal bones, and a prominence forms over the carpus and the dorsum of the hand, which alarms the patient but is of no consequence. It is known as Gubler's tumor.

THE SKIN. The *skin* of the hand need not concern us, save as estimated in connection with the skin of the rest of the body. It is smooth or rough, dry and harsh, moist and warm, under the same circumstances that affect the skin generally. In rheumatoid arthritis it has been particularly described as peculiar. Both the dorsal surface and the palm are moist and very soft, and the former dotted with freckles.

The *swellings* of the hand, inflammatory or œdematous, do not differ from swellings in other portions of the body, whether the joints are affected or the subcutaneous connective tissue, except in the cases previously mentioned. (See Skin.)

Fingers. In gout and rheumatism the *fingers* present changes. The swelling of the joints in each condition cannot well be distinguished. In gout, tophi are likely to be present in the joints or along the tendons, on account of great accumulation of urate of soda. They are more prominent on the dorsal surface of the joints, and sometimes break through the skin, so that the "chalk-like" concretion exudes. It was said by Sir Thomas Watson that a gouty subject under his care utilized his joints in keeping tally while playing cards.

Heberden's Nodosities. The term "end-joint arthritis" is also applied to these nodes. The nodules develop gradually at the sides of the distal phalanges. The subjects may be in good health, or may have had attacks of gout, or have suffered from acid dyspepsia. At first the joints may be a little swollen and tender. The swelling and tenderness may occur in paroxysms, and with each paroxysm the size may be larger than at the preceding paroxysm. The tubercles are seen at the side of the dorsal surface of the second phalanx, the corresponding cartilage becomes soft, the ends of the bone may be eburnated. A moderate ankylosis takes place. They are often considered of good prognostic omen; it is even said they are a sign of longevity. It is certain that the large joints are not involved when these nodosities are present.

In acromegalia and pulmonary osteo-arthropathy the state of the fingers has been described. Heberden's nodes and Haygarth's nodosities have also been noted. The tips of the fingers are bulbous, or *club-shaped*, in cases of phthisis and in other forms of chronic lung disease and in chronic heart disease. It is most common, however, in bronchiectasis and phthisis. The clubbing is associated with changes in the nails (see *infra*). In addition to the nodosities above mentioned, extra-articular tophi which develop in the course of gout must be referred to.

Deviations in the Position and Shape of the Fingers. Changes in the shape of the fingers occur as described in connection with the changes in the shape of the hands. The eversion in rheumatoid arthritis is characteristic of that affection, but deviations due to abnormal flexion or extension produce the most marked changes. Flexion of the first phalanx of the little finger is due to contraction of the palmar fascia, or to paralysis of the common extensor on account of disease of the musculo-spiral nerve.

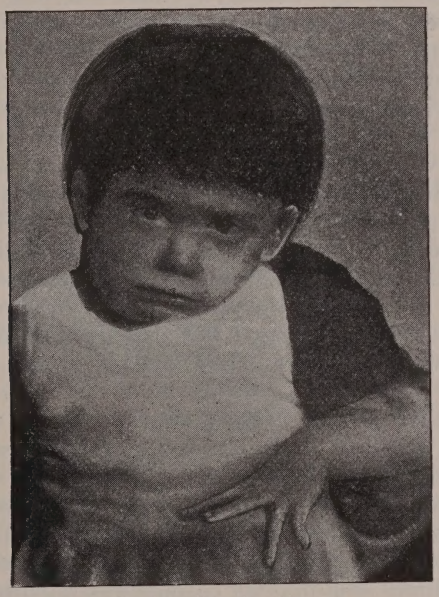
Contraction of the fascia of the hand, on account of which the little and ring fingers are flexed in more or less degree, is frequently seen, and may be an indication of gouty diathesis. It is certain that these contractions are seen in several members or generations of a family in which gout is prevalent. It is called Dupuytren's contraction.

Abnormal *extension* is usually very marked. When the middle phalangeal joint is affected the hyper-extension is due to disease of the median nerve, on account of which there is paralysis of the flexor sublimis; there is hyper-extension of the distal joints, with paralysis of the flexor profundus muscles from disease of the median and ulnar nerves. In *main-en-griffe*, previously described, there is extension of the proximal phalanx with extreme flexion at the same time of the two distal phalanges, due to contraction of the long extensor and of the flexors. Contractions due to chorea or to central lesions, as post-hemiplegic contractions, will

be considered under special diagnosis. It is thus seen that the peculiar combined extension and flexion, causing abnormal shape of hands and fingers, is due either to (1) local joint inflammation (subluxations); (2) local neuritis and paralysis; (3) progressive (spinal) muscular atrophy; (4) idiopathic muscular atrophy, rarely.

Athetosis is a peculiar spasmodic affection of the fingers and toes, often hereditary, and nearly always associated with imbecility or some intra-cranial lesion. It may be unilateral or bilateral. There is contraction or paralysis of the affected limb. The muscles may be atrophied

FIG. 14.



Case of athetosis. (GRAY.)

or hypertrophied. The characteristic feature is the slow, wavy, and gradual movements, which are continuous. The fingers constantly tend to pronate, but the toes do not separate.

Tropho-neurosis of Fingers. Changes in the appearance of the extremities and nutritive changes are seen, due to diseases of nerves which control nutrition.

THE CIRCULATION. RAYNAUD'S DISEASE. *Local asphyxia.* In certain vasomotor affections the hand or fingers become pale, intensely cold, are the seat of numbness, and are without sensation. The term "*dead fingers*" graphically describes the appearance. The pallor usually comes on suddenly, and continues for a short or long period of time. In some instances it occurs in distinct paroxysms. The disappearance of the pallor is marked by a gradual return of warmth to the part and change in color to a livid red, dark blue, or even blackish hue. In some cases the lividity becomes so intense that gangrene in small superficial spots, or involving the whole finger, ensues. Pain may or may not be present, but is not increased when the hand hangs down.

The tip of the nose and the lobe of the ear may be affected. The sensation to touch is markedly lessened. *Raynaud's disease*, for this is the affection under consideration, occurs usually in ill-nourished subjects or after an acute disease, as typhoid fever.

Erythromelalgia. Local changes in *color* are due to peripheral neuritis or neuritis of the terminal endings of the nerves.

Erythromelalgia is characterized by redness of the surface with increased temperature; it is usually seen in the extremities and limited to the distribution of nerve areas. It is worse in summer, increased by heat, and aggravated when the extremity is dependent or pressed upon. The redness is attended by burning, by most extreme local discomfort, in which all sorts of sensations are described. Tearing of the finger-nails, pulling or pricking of the skin, twistings of thousands of needles, and other painful sensations have been used to describe the suffering. I know of no peripheral pain which is the source of greater agony.

Glossy Skin is seen after nerve injuries and neuritis, and in central affections in which the trophic nerves are involved. The skin is shiny, smooth, drawn very tightly over the surface, and sometimes atrophied. Red and pale mottling may be seen. The surface is free from hair. Burning pain precedes and accompanies the change. (See Nails.) In addition to the gangrene previously noted, other pronounced trophic changes are seen in the extremities. *Perforating ulcer of the foot* is an example of such change; it is usually seen in affections of the general nervous system, such as tabes dorsalis.

The Nails. The Shape. The appearance of the nails gives information as to the duration of some diseases or of convalescence, and to the local interference with the nutrition of the parts. Thus, curving of the nails, with the club shape of the finger-ends, occurs only in chronic diseases, as in cases of phthisis or emphysema, or in chronic cardiac disease and aneurism. In the latter it is sometimes found on one hand only. It is sometimes seen in other chronic wasting diseases. The nails may curve transversely or longitudinally. When transversely the appearance is like that of a filbert, and when longitudinally they are said to be incurvated. This change in shape may occur without clubbing of the fingers. The shape is altered in acromegalia and pulmonary osteo-arthropathy.

Color. White marks on the surface are usually seen after an illness, and may indicate the length of time since the illness occurred. The marks develop at the root of the nail, and their position denotes the time that has elapsed since convalescence set in. If they are seen half-way up the nails, convalescence is probably of three months' duration. We get a good idea of the condition of the blood in the capillaries from the appearance of the tissue under the nails. If there is anæmia, pressure on the finger-tips will drive the blood from the capillaries. Stephen Mackenzie's rule, that if such pressure completely empties the vessels so that they are pale it indicates that the globular richness of the blood is reduced one-half, is a fair and quick test to indicate the degree of anæmia. The purplish and bluish-black discoloration of *cyanosis* previously referred to is first seen under the nails. Sometimes the *capillaries pulsate*, and this pulsation is more

visible in the nails than in other parts of the body, except the retina. It occurs in the course of aortic regurgitation.

Nutritive Changes. The nails undergo chronic inflammation with destruction in various skin affections, and the matrix is the seat of acute inflammation in onychia. Onychia may be simple or syphilitic in its nature. Its presence may explain the course of obscure nervous phenomena. It may be limited to a simple inflammation, or with subsequent loss of the nail and further ulceration going on to necrosis.

Deformity of the nails (toe) occurs in acute and chronic myelitis. In *locomotor ataxia* the nails fall out. In *neuritis* the trophic change is marked; the growth is arrested and the nail becomes dark and brittle and curved in its long axis, while lateral arching takes place. The cutis underneath thickens and the skin at the base retracts. The fingers may be clubbed. When growth is resumed a roughened distinct line of demarcation is seen. In some cases they become dry, scaly, and cracked, or atrophy entirely. In hemiplegia from cerebral apoplexy the growth is arrested on the paralyzed side. This is tested by staining the nails of the two hands at the same level with nitric acid; the relative position of the stain upon corresponding nails of the two hands will show whether there has been growth or not. The return of functional power is indicated by renewed growth.

The Feet. The feet and ankles are examined to determine the color, the temperature, the occurrence of swelling (œdema), and fixation. Pain in the feet has been referred to; œdema has also been discussed. The changes in color are allied to the same in the hand if bilateral.

Cold Hands and Feet. Changes in the temperature of the extremities are frequently complained of by patients, and on examination we find it actually reduced. It is a common and often a serious complaint. It is natural to expect a peripheral coldness when the central organ of circulation is weakened. In the final hours preceding death coldness takes place. But in organic disease of the heart, with impairment of the circulation, we also see it. It is a common vasomotor condition in states of nervousness independent of hysteria. A visit to a physician, excitement from any cause, is likely to be attended by coldness of the hands and feet. Under these circumstances the extremities are bathed in perspiration of a cold and clammy character. In endarteritic changes occurring in the aged, cold hands and feet frequently occur. They are an index of the state of the peripheral circulation, and may explain the cause of many of the symptoms which so frequently accompany it.

In gout and rheumatism, and in morbid conditions in which poison circulating throughout the body irritates peripheral and vasomotor nerves, cold hands and feet are likely to be annoying. Patients with forms of indigestion, as well as the above-mentioned states, complain of this affection constantly.

Changes of *sensation* in the skin of the extremities will not be considered in this section. The alterations are so bound up in diseases of the nerves that an account of their diagnostic features will be considered

in the chapters devoted to these diseases. It is sufficient to state that anæsthesia is seen in local areas and from causes limited to the skin in morphœa, in the anæsthetic form of leprosy, and in certain ischæmic states (urticaria). The loss of tactile sensibility accompanies it. Hyperæsthesia and paræsthesia occur with various local affections, but are without diagnostic significance, except in nervous diseases.

The Lymphatic Glands. (See Neck.) Examination of the condition of the lymphatic glands leads to information which may be of diagnostic value. They may be enlarged in infectious diseases, notably *syphilis*. The *post-cervical* glands, the *epitrochlear* glands, and lymphatic glands in other portions of the body, point to this condition. In the former localities the enlargement is of great diagnostic importance, as it is less likely to have been caused by other conditions. The enlarged glands that suppurate in local areas do not here concern us. *Inguinal and axillary enlargement.* With or without suppuration, enlargement always points to an irritation or lymphatic invasion in the area which the affected lymphatic gland drains. When in the groin, the feet are affected, and when in the axillæ, the hands. Great enlargement in either situation causes œdema of the corresponding extremity, if the veins are pressed upon. The axillary glands are early affected and enlarged in mammary cancer. The breast should always be examined in œdema of the arm.

The supra-clavicular glands. The only local enlargement that is of special diagnostic significance is that which is seen above the clavicle on the left side. The glands are enlarged and indurated, and may cause pressure symptoms. With other symptoms they point to the occurrence of carcinoma of the stomach. Indeed there are cases of this disease in which the general symptoms of carcinoma alone are present. Local symptoms are wanting, and the locality of the cancer cannot be made out. The enlarged glands above the clavicle are a pretty sure indication that the disease is seated in the stomach. Enlargement in this locality is probably due to transmission of the infection along the thoracic duct and the associated glands. *The cervical and sub-maxillary glands* The enlargement of the sub-maxillary and cervical glands points to affections of the mouth and throat, and disease of the jaw and teeth. It is caused particularly by infectious disorders in these localities.

The glands are enlarged in *adenitis, tuberculosis, Hodgkin's disease, leucocythæmia, sarcoma,* and *cancer.* The moderate enlargement of syphilis and local enlargement from irritation in the area of lymph drainage has been spoken of. *Adenitis* is usually local. The gland is tender; the connective tissue around it is affected. There is local heat and pain. At first the gland is hard, then softens in the centre, and finally exhibits fluctuation. In tuberculosis more than one gland is affected. Usually the disease is bilateral (as in the neck). At first the glands are isolated. Later they become matted. The local symptoms are not marked, and are very indolent. The course is slow. Thick, cheesy pus is removed, which may contain tubercle bacilli. It always causes tuberculosis when inoculated in lower animals. Fever and "decline" occur later, but

often not until other structures, as the lungs, are infected. (See Hodgkin's Disease, and Leucocythæmia.)

Lymphangitis or angioleucitis. The streaked redness over the surface of the skin, with tenderness along the course of the lymphatics and œdema below, characteristic of inflammation of the lymphatic vessels, need not be further mentioned. The characteristic appearances that are seen in *elephantiasis*, associated with a change in the urine known as chyluria, with or without lymph scrotum, point in an unerring manner to the occurrence of the affection due to the *filaria sanguinis hominis.*

Muscles. The Nutrition. The nutrition of the *muscles* is observed with the hand of the examiner when the muscles are made to relax and contract alternately. (See *Vierordt.*) Comparison of corresponding muscles of the two sides is made. Change is observed more accurately by measurement of the limbs at corresponding situations. The muscles may *atrophy* or *hypertrophy.* Either condition may be local, unilateral, or general and bilateral. Atrophy is due to several causes: 1. The atrophy of disuse. 2. The atrophy of degeneration. It occurs in lesions of the motor path, cortical, medullary or spinal, or in neuritis. (See Nervous Diseases.) 3. Myopathic atrophy.

Atrophy. Atrophy of the muscles from disuse or disease of the muscle must be distinguished from atrophy due to disease of the nerves (neuritis) and degeneration of motor nerves and ganglia. The former is also known as the atrophy of inactivity. The muscles are slightly diminished in volume. The atrophy takes place very slowly. It supervenes in cases of paralysis. It occurs in joint disease on account of which the limb or a portion of it has been kept at rest. It occurs also in joint disease from reflex influences. The electrical sensibilities of the muscles are qualitative and unchanged.

General Atrophy. In cachexias in addition to atrophy of the tissues the muscles undergo atrophy. Even in nervous diseases the atrophy of the muscles due to the disease markedly increases when general wasting takes place.

Myopathic Atrophy. In this form of atrophy the muscle is subject to disease. It diminishes in volume, and finally becomes completely shrunken. Complete paralysis rarely ensues, but reaction of degeneration cannot be determined. This form of atrophy occurs in idiopathic or progressive muscular atrophy.

Idiopathic Muscular Atrophy. In this affection muscular wasting takes place with or without initial hypertrophy. Three forms are seen:

1. Atrophy, with Pseudo-Hypertrophy. It usually begins in children, and is often of congenital origin, transmitted through the mother. It is first noticed just as the child is learning to walk. The extensors of the leg, the glutei, the lumbar muscles, the deltoids, and the triceps and infraspinati muscles are involved, but the primary change takes place in the muscles of the calves. The muscles of the face, neck, and forearm are not usually affected in this form; the muscles of the hand are not involved. While hypertrophy progresses in certain muscles, others waste. The calves may hypertrophy, for instance, while

the extensors of the leg are wasting and become weak. Attitude and gait are characteristic (see page 61). The patient stands erect with the legs far apart, the shoulders thrown back, the spine curved, and the abdomen prominent. The waddling gait is characteristic, and the method of getting up from the floor is pathognomonic. The course of the disease is slow, wasting follows the hypertrophy, but the weakness is greatest in the primary atrophied groups. Contractures and distortions of the spine and bones of the leg take place.

2. Primary Atrophy. This is likewise congenital, and occurs in early life. It is divided into different types according to the groups of muscles that are affected. The same process occurs as in the former, except that pseudo-hypertrophy is not primary. There may be several forms in different members of the same family.

a. The juvenile form of Erb. The upper arm and shoulder and the thigh muscles are first involved. Later the muscles of the gluteal region and calf may be enlarged and hard. The back muscles are gradually affected, and the attitude previously mentioned is taken. The reaction of degeneration is not present. In addition, the *infantile type* first described by Duchenne, or the *fascio-scapulo-humeral type* is seen. Erb's form begins about puberty. The other forms begin in childhood, but may be delayed. The face is involved; it is expressionless, so that in laughing the muscles move slowly; and the lips cannot be employed in whistling, as they are thick and everted. The eyes remain partially open. The muscles of the group waste; later the thighs become involved. Erb has given a useful test to determine the strength of the shoulder and girdle muscles. When the child is lifted by the armpits, if the scapulo-humeral groups are weak the shoulders are forced up to the child's ears without resistance.

Diagnostic Features. The disease is characterized by gradual progression of the wasting and weakness in various groups of muscles not especially related. We never see wasting of the intrinsic muscles of the hand, as in the spinal forms of muscular atrophy, or of the tongue, pharynx, larynx, and eye. Electrical irritability is lessened, and reaction of degeneration is not present. Fibrillary twitching is not seen. Sensation is not affected. The reflexes are diminished, and later may be lost. The sphincters are not involved; deformities about joints or of the spinal column may occur. A *peroneal type* of muscular atrophy has been described by Charcot. The extensors of the great toe and afterward the common extensors and peronei muscles are affected; club-foot results. The muscles of the thigh may become involved later. When it occurs in childhood the disease gradually progresses to the upper extremities. The muscles of the hand become affected, in which it differs from other forms of muscular atrophy. The thenar, hypothenar, and interossei muscles are symmetrically involved, producing the claw-hand. Unlike the other forms of atrophy embraced under this heading, disturbances of sensation have been described, and in addition pain, fibrillary contractions, and vasomotor changes. The reactions of degeneration may be present. It is thought by competent observers that it is simply a form of neuritis.

The *diagnosis* of idiopathic muscular atrophy is not difficult, if the

above-mentioned facts are borne in mind. The occurrence in family groups is important to remember in the diagnosis. In *cerebral atrophy* there is primary loss of power. In *chronic anterior poliomyelitis* (*spinal atrophy*), atrophy begins in the muscles of the hands first; in both the simple and the spastic form there are reactions of degeneration, fibrillary twitching and increase in the reflexes, and in the latter spastic contraction of the legs. The myopathies occur early in life and are hereditary.

In *neuritis* the paralysis is proportionately greater than the atrophy. Sensory symptoms are often present. The cause is distinct. There is no family history.

Hypertrophy of the Muscles. Hypertrophy of individual muscles occurs from overuse, and is seen when one extremity or one portion of the trunk is used, and comparatively in excess, in the daily pursuit. General hypertrophy of muscles occurs in Thomsen's disease. True hypertrophy is recognized by increased volume, great hardness and increased vigor of the muscle.

Pseudo-Hypertrophy (see under Muscular Atrophy) is associated with increased volume of muscle, but diminished power.

Thomsen's Disease. (*Myotonia congenita.*) This is an hereditary disease and may occur in several generations of a family. Tonic cramps take place in the muscles when an attempt is made to make voluntary movements. The disease begins in childhood, rarely after puberty. The muscles become rigid and fixed when an attempt is made to move them. The lack of voluntary control of the muscle is seen in slow contraction and relaxation when voluntary efforts are made. The rigidity may wear off and the limb can then be used. It is particularly noticeable when walking is attempted; as the leg is advanced slowly it may remain stiff for a second or two, but after it becomes limber the patient can walk for hours. If he stops walking the same difficulty is experienced when it is resumed. Both the arms and the legs are affected. Patients are usually well nourished, however. There are no atrophies. The muscles are irritable, so that mechanical stimulus or pressure causes tonic contraction. Movement and cold aggravate it. Sensation and reflexes are not affected, and there is no evidence of disease of the cerebro-spinal system, save the occurrence of hypochondriasis in some cases. The myotonic reaction described by Erb is induced. (See electrical diagnosis—Diseases of the Nerves.)

Paramyoclonus Multiplex. In this affection there is clonic contraction of the muscles. It is usually confined to the extremities, and occurs in paroxysms. It may have been caused by sudden twitching or violent motion. The clonic spasms at first do not interfere with the patient's occupation, but gradually increase. Both legs are affected and the contractions vary from 50 to 150 in the minute. The contractions may be rhythmical. In severe cases the muscles of the back and abdomen contract violently. Tremors of the muscles may be present in the intervals. (For spasm, tremor, contraction, etc., see Nervous Diseases.)

Myositis. *Inflammation of the muscles.* (For changes in the muscles due to trichinosis, see that disease.) In inflammation of the muscle there is pain, swelling, and loss of power. In universal myositis the inflammation begins in the muscles of the lower extremities and gradually involves other muscles of the body. The muscles are swollen, hard, and painful on pressure. Atrophy supervenes in groups of muscles. The progress is gradual, and death ensues from involvement of the respiratory muscles. The muscles may become stiff and more or less rigid. Local œdema of the skin over the muscle occurs.

The three cardinal symptoms that attend the disease as described by Loenfeld are: (1) Swelling of the extremities due to subcutaneous œdema and swelling of the muscle, on account of which there is disturbance of function; (2) extension to the muscles of respiration and deglutition; (3) a more or less extensive eruption. The latter is erythematous, its distribution is usually general but irregular, and may be followed by pigmentation. The disease must not be confounded with trichinosis. In the latter, examination of a small portion of muscle discloses the trichinæ. *Progressive ossification* of the muscles is rare. The muscle tissue undergoes gradual ossification, either in localized spots or in widespread areas. Inflammation of the muscle precedes the ossification. As the inflammatory swelling subsides, the muscles become hard and are gradually converted into bony tissue. The disease lasts over a great number of years.

Raymond's Table of Atrophies.

- **Circumscribed atrophies**
  - Atrophy from compression.
  - Atrophy in inflammatory conditions (pleurisy, joint disease, etc.)
  - Atrophy from injury or inflammation of individual nerves.
- **Progressive atrophies**
  - Progressive spinal muscular atrophy; type Aran-Duchenne.
  - Progressive myopathic atrophy
    - Pseudo-hypertrophic muscular paralysis.
    - Type Leyden-Möbius.
    - Type Zimmerlin.
    - Type Erb.
    - Type Landouzy-Déjérine.
    - Type Charcot-Marie.
- **Diffuse atrophies**
  - Anterior poliomyelitis
    - Infantile form.
    - Acute of adults: spinal paralysis, with rapid course and curable (Landouzy-Déjérine); subacute and chronic form; chronic mixed form (Erb); diffuse subacute, general spinal paralysis (Duchenne).
  - Syringomyelia.
- **Facial hemiatrophy**
  - Multiple neuritis (amyotrophic form)
    - Lead paralysis.
    - Leprous neuritis.
    - Alcoholic neuritis.
- **Muscular atrophies of cerebral origin**
  - With secondary degeneration involving the anterior cornua.
  - Without secondary degeneration involving the anterior cornua.
- **Muscular atrophy in hysteria**
- **Muscular atrophy from systemic disease of the cord**
  - Amyotrophic sclerosis.
  - Glosso-labio-laryngeal paralysis.
- **Atrophy complicating other disease of the cord**
  - Atrophy in myelitis.
  - Atrophy in compression of the cord.
  - Atrophy in multiple sclerosis.
  - Atrophy in tabes dorsalis.

The Bones. The examination is made by inspection and palpation. The student should familiarize himself with the shape of the bones and the seat of normal tuberosities. He should learn the movements of the spine and its position in health. Examination is usually made to determine their position and shape, and, in addition, to ascertain the presence of local changes.

Local Changes. Changes in the bone that appertain to general changes of the skeleton have been referred to. Local examination of the bones, however, is of the greatest importance. The discovery of a slight change may lead to the recognition of a grave general process. We examine for local inflammation and the presence of nodes. Simple local inflammation or *periostitis* may be due to syphilis, and is recognized by local pain, swelling, and slight œdema. It may be diffuse. It is seen most frequently on the tibia, sternum, and clavicle. *Nodes* form on various portions of the skeleton, but are most frequently seen on the skull, and of this region the forehead; or on the shafts of the long bones, preferably the tibia, ulna, and clavicles. They are usually multiple or bilateral. They are not so hard and dense as exostoses. The latter are situated on the outer aspects of the bone and in relation to tendons or muscles which are characterized by vigor of action.

As an illustration of the importance of recognizing nodes the writer has seen a case of persistent headache, the true nature of which was only ascertained by finding a small node on the skull. The headache had been of long (five years) duration, and treatment for it sought in many countries.

Tenderness of the sternum upon pressure is often of diagnostic significance and usually indicative of syphilis. The pain and tenderness just noted, however, must not be confounded with local tenderness due to necrosis which often arises in the convalescence of fevers, notably typhoid.

Position and Shape. The peculiar position (falling downward) assumed by the *scapula* in paralysis of the serratus magnus is diagnostic of that affection, and indicates disease of the posterior thoracic nerve. In examination of the *clavicles* fractures must not be taken for disease of the bone, of which *rickets* is the most common. The examination of the *spinal column* is of the greatest importance. (See Spinal Joints, next chapter.) It is not within the province of this work to include the study of the diseases of the spinal column due to caries from tuberculosis. Observation of all patients is, however, not complete without noting the movability of the spine and the presence or absence of curvature. I refer to the curvature due to weakness of groups of spinal muscles. Without doubt functional disorders of the gastro-intestinal tract and of the uterus are intensified by the presence of curvature, which leads to deformity of the body and hence the assuming of improper positions when sitting or walking. To recognize the lateral or anterior curvature is to be able to put the patient on lines of treatment which otherwise would not be followed, but without which weak muscles, improper aëration of blood, and sluggish circulation would persist. The occurrence of pain in the distribution of nerves, or at their termini, is often due to spiral caries pressing on them as they pass through the foramina. The most noticeable is the pain about the umbilicus in children, due to Pott's disease.

The bones and cartilages connected with the thorax will be considered under Diseases of the Lungs.

Osteomyelitis. The occurrence of high fever, with or without chills but usually with pyæmic symptoms, should conduct inquiry to the bones, which must be examined carefully. A spot of tenderness followed subsequently by local redness and swelling—on the tibia, for instance—would indicate the seat of suppuration in *osteomyelitis.*

The Joints. By *inspection* and *palpation*, changes in the joints are observed which are of great significance in the recognition of various morbid processes.

Inspection. The *size*, *shape*, and *color* are observed, and the *position* assumed noted. In addition, the movability of the joint is investigated. The nature of the joint affection is learned further by knowledge as to the number of joints affected, the limitation to large or small joints, the occurrence of metastasis. Polyarticular inflammation of small joints points to rheumatoid arthritis; of large joints, to rheumatism; monarticular inflammation of small joints, to gout; of large joints, to gonorrhœal rheumatism or pyæmia; sudden flitting from one joint to another is characteristic of rheumatism.

The Size and Shape. The joints may be enlarged. The enlargement may be due to infiltration of the tissues about the joints, to effusion within the joints, serous or purulent, or inflammation of the ends of the bones. 1. When the enlargement is due to infiltration about the joint, the tissues are previously thickened, as indicated by palpation, and the outline of the joint is changed. The normal contour is lost entirely, and, instead, a globular swelling beginning above, and extending below the joint is seen. 2. When enlargement is due to effusion it may be detected by palpation, by which fluctuation is secured. This is particularly so in the large joints. If it is the knee, the patella will float. The effusion changes the normal contour, but in the earlier stages may cause local swellings at parts where the synovial sacs are near the surface; hence, at the articulation of the tibia and fibula with the tarsus on the inner and outer side, a baggy swelling is observed. At the knee the swelling is on each side above or below the patella. Where effusion is great the joint becomes immobile, and may be flexed on account of distention of the sac. 3. When enlargement of the joints is due to hypertrophy of the bones, the latter are thickened and very hard. There may or may not be, and usually is not, fixation, and movement is but moderately interfered with.

Changes in the *outline* of the joint are seen in addition to the above in rheumatoid arthritis. The loss of the cartilaginous substance of the joint, with the secondary osteophytic changes, cause distortion, so that in the case of the small joints of the finger subluxation is seen; similar subluxations are seen in larger joints likewise. The ends of the phalangeal bones are thickened. Change in the *color* is usually noted in inflammations. Its surface is bright red or dusky.

The *position* assumed is of diagnostic importance. Flexion of the limb of the affected joint occurs in overdistention. In rheumatoid arthritis there is subluxation. Immobility is observed. (See Palpation.)

PALPATION. The results of inspection are confirmed. 1. The movability of the joint is learned. In inflammation it is attended by pain; movement is inhibited. A reflex spasm takes place if osteitis and cartilage destruction are present. The spasm prevents movement. In effusion there is less or even no movement whatever. In rheumatoid arthritis movement is prevented by the osteophytic growths which surround the joint.

By palpation fluctuation is detected, pointing to swelling on account of effusion. Pitting on pressure is found in suppuration of the joint.

In rheumatoid and other destructive diseases, a crepitus or grating sensation is observed.

The *subjective symptoms* of joint affections are worthy of note. *Pain* is the most prominent. This may be spontaneous or may arise upon pressure, or be due to attempts at movement. Spontaneous pain with tenderness is more pronounced in rheumatic and gouty inflammations of the joints. The pain is usually worse at night. This is particularly the case in tuberculous joints, and is due to removal of the apprehensive spasm of the muscles whereby the joints had been protected.

The *pain* in the joints must not be confounded with the pain that attends local or multiple neuritis. I have seen the pains of neuritis attributed to rheumatism of the phalanges, tarsus, and ankle, until paralysis of the extensors took place. I have seen the pain of neuritis of the circumflex taken for shoulder-joint disease. Multiple neuritis is attended by pains that may be located in the joints by the patient; but whether local or general neuritis, the joints are never swollen, tender, or painful on movement by the hand.

THE JOINTS OF RHACHITIS. (See under Rhachitis.)

THE JOINTS OF OSTEO-ARTHRITIS. (See under Skeleton.)

THE JOINT OF SYNOVITIS. The inflammation is recognized by pain, heat, redness, and swelling. Effusion is present, physical signs of which are readily elicited. It may be due to traumatism, but the inflammations due to internal morbid processes concern us. The most common are tuberculosis, pyæmia, and gonorrhœal infection when single joints are affected. A mild degree of inflammation may be limited to one joint in subacute rheumatism. In tuberculosis the joint is swollen and the neighboring tissues œdematous. Effusion may be detected. There is fever. The hip, the knee, the elbow, the wrist and the ankle are most frequently affected. Cheesy material may be withdrawn by tapping. Destruction ultimately takes place, with subluxations and subsequent fixation of the joint. With the fever, wasting and other signs of tuberculosis, and the occurrence of tuberculosis in some other portions of the body, point to the true nature of the affection. The tuberculous process may be limited to the affected joint, or secondary tuberculosis may supervene.

THE JOINT OF GONORRHŒAL RHEUMATISM. Signs of acute or subacute inflammation are present with œdema and effusion. The patient is a male in whom an acute or chronic urethral discharge is found. The pain is worse at night. The process is of long duration. Metastasis does not take place. Destruction rarely occurs, but ankylosis may follow. General pyæmic symptoms may ensue, and ulcerative endocarditis

supervene. There is entire absence of heart symptoms from simple endocarditis. The general and local signs of rheumatism or of a rheumatic diathesis, and changes in the urine, skin eruptions, cardiac lesions, etc., are wanting.

The Joint of Gout. Any joint may be affected, but the typical gouty inflammation is seen in the metacarpo-phalangeal joint of the great toe—the ball of the toe There is great swelling, intense redness, enlargement of the veins, and œdema. There may be some effusion; it results in chronic inflammation and enlargement of the joint. Tophi about the joints are observed. Agonizing pain occurs, and is worse at night. Fever attends the process. The attack is of short duration, and may be followed or attended by acute gouty inflammation of other structures, or vascular and renal changes associated with this general morbid process.

The Joint of Rheumatism. It is swollen, painful both spontaneously and on movement, and there may be some redness of the surface. Other joints are soon attacked, with subsidence of the symptoms in the original joint. The large joints are usually affected. It may be limited to one side or may affect both. Secondary or concurrent cardiac inflammations may be noted. High fever and acid sweats attend the process, which is common in both sexes in childhood and early adult life. Other evidences of the rheumatic diathesis and the history of previous attacks point to the true nature of the joint swelling.

The Joint of Rheumatoid Arthritis. There may be simple chronic inflammation with acute exacerbations, or prolonged subacute inflammation. The small joints are affected first, as the phalanges. They are swollen and the adjacent structures infiltrated. At first there may be, particularly with each exacerbation, some effusion. Later the cartilages are eroded, and crepitus and grating are detected on palpation. Subluxation with great deformity ensues, followed by complete fixation of the joint. The crepitation may be detected along the sheaths of the tendon. Osteophytes develop. The skin over the surface becomes glossy, and the affected hands are covered with freckles. Occurring in early adult life, usually in females with marked anæmia and secondary wasting of the muscles without heart lesion or general indications of rheumatic or gouty diathesis, the true nature of the swelling is early recognized. (See Rheumatoid Arthritis—Extremities.)

The Tabetic Joint. In forms of nervous diseases, particularly in sclerosis of the posterior columns, secondary joint involvement sometimes follows. The change in the large joints is first preceded by pain, stiffness, and inability to use them. Gradually nutritive changes take place. At first there is boggy swelling. The cartilages become eroded, the heads of the bone waste, the ligaments ossify, and irregular bony growths project. Wasting of the head of the femur is followed by dislocation. Sometimes an effusion takes place in the joints, and there may be peri-articular œdema. The large joints are most commonly affected—the knee, hip, ankle, and elbow. Injury excites the abnormal trophic process. When the tarsal bones and the articulations are affected the foot becomes flat, and the tarsal and metatarsal articulation and the tarsal bones project forward or backward. This is called the tabetic foot.

The Joint of Hysteria. Symptoms referable to the joints are sometimes complained of in hysteria. Pain and fixation of the joint are complained of. The joint rarely undergoes organic changes, but sometimes a plastic infiltration of the connective tissue outside of the capsule does occur. The hysterical nature of the pain and immobility are recognized by the absence of a cause for joint lesion, the absence of fluctuation, or of signs due to erosion, by the association of the local symptoms with the phenomena of hysteria, but more particularly by the fact that contraction and even wasting precede the joint symptoms. In true affections of the joint both occur after the joint has become diseased; in hysteria muscular contraction will take place first.

The knee is the joint usually affected. Care must be taken not to be deceived by local vasomotor changes of hysterical origin which may be observed over the surface of the joint. This local increased temperature is not associated with general fever, however, while the vasomotor changes indicated by swelling of the skin, increased tension, and the shining appearance, with increased sensibility, are not persistent, but occur once or twice in the twenty-four hours. In a remarkable case of Mitchell's the local vasomotor change took place at night. The temperature of the knee which was affected would increase three or four degrees, while the pulse remained at 80. The local symptoms of heat, redness, swelling, tension, and increased pain would pass away by three o'clock in the morning. The fact that the same symptoms could be brought about by handling the knee, or by pressure upon the patella, pointed to its vasomotor origin.

In joint cases, as was the case with the one just noted, a study of the reflexes is made. The reflexes do not change, electrical reactions are normal, although there may be atrophy from disuse, but not to the degree that occurs in organic disease. The muscles were contracted, but, as previously noted, the contracture was primarily a relaxation, which took place if the tension was removed. Concerning these vasomotor changes, Sir James Paget's expression, "A joint which is cold by day and hot by night is not an inflamed joint," is a safe guide to the recognition of a joint affection. When the joint becomes hysterical after injury it is most difficult accurately to ascertain its true nature.

Special Joints. The three joints that should concern the student more particularly are the shoulder, hip, and knee. When symptoms are referred to either of these joints they should not be passed over lightly. Grave consequences have followed attributing inflammation of the hip-joint to rheumatism when it was of tuberculous origin. But not only has hip-joint disease been mistaken for rheumatism, but the mistake has been made of considering the process to be going on in the knee instead of the hip. This has arisen because there is often flexion of the leg and because pain is so often referred to the knee-joint.

On the other hand, cases of hip-joint disease have been mistaken for suppuration in the pelvis or in the iliac fossa. Typhlitis or appendicitis has frequently been mistaken for hip-joint disease.

In the shoulder-joint the danger is in confounding neuritis of the circumflex nerve and consequent paralysis of the deltoid with affections of the joint. If it takes place about the joint and there is inability to move

it upon the part of the patient, it is still readily moved by the physician, and the physical signs of joint inflammation are wanting when sought for.

Method of Examination. In the examination of bones and joints, particularly the spinal column, it is necessary that the patient should be stripped, and in addition to noting the movements in the upright, or semi-upright posture, as well as positions assumed in each, the position of the trunk and of the joints should be examined with the patient lying down. A hard, smooth surface should be selected. In this manner deformities, changes in the length of the bone, and abnormal posture can be carefully observed. In addition we must note muscular wasting, the occurrence of local tenderness and swelling, changes in the length of the bones, changes in the movements of the joints, and loss of other functional activity causing lameness or joint disability.

Diagnostic Significance. The diagnostic significance of the distribution of the lesion in joint affections is of great importance. Lesions may be unilateral or bilateral, and may be symmetrical or asymmetrical. They may be limited to the small joints or to the large joints alone. Bilateral joint lesions are characteristic of rheumatoid arthritis. In such disease, moreover, the small joints are particularly involved. In gout the small joints are primarily affected, though the large joints may become affected secondarily. In rheumatism, on the other hand, larger joints are first involved. This affection is particularly specialized by the occurrence of asymmetrical inflammation in many joints, the irregularity of its distribution and the fugaceous nature of the joint affection. Monarticular inflammation is seen in gonorrhœal rheumatism. In pyæmia the large joints are involved. The range of movement and the evidences determined by palpation are not of marked diagnostic significance. In all joint affections the movement is limited and painful, upon both active and passive movement.

CHAPTER IV.

BACTERIOLOGICAL DIAGNOSIS.

Causal relation of bacteria to disease. Koch's laws; value in diagnosis. Method of research: Microscopical examination, cultivation, inoculation. Essentials in technique.—*Bacteria:* Saprophytes, parasites, pathogenic, non-pathogenic, aërobic, anaërobic, facultative anaërobic. Morphology: micrococci, bacilli, spirilla. —*Micrococci.* Morphology: Form and size. Reproduction, fission; grouping. Biological characters: Non-motile. Pigment production. Liquefaction of gelatin. Production of acids. Toxic ptomaïnes and toxalbumins —*Bacilli.* Morphology: Form and size. Reproduction, fission, spores; grouping. Biological characters: Motility. Pigment production. Liquefaction of gelatin. Production of acids. Putrefaction, fermentation.—*Spirilla.* Morphology: Form and size. Reproduction, fission; grouping. Biological characters: Motility. Pigment production. Liquefaction of gelatin. Production of acids and fermentation wanting.—*Method of research:* Blood, discharges, exudations; mode of collection. Apparatus. Preparation of apparatus. Sterilization. Microscopical examination: Technique, cover-glass preparations. Methods of staining; spores. "Hanging drop."—Cultivation of micro-organisms. Culture media. Tube and plate cultures. Smear and stab cultures.—Inoculation of animals.—Special bacteriological diagnosis.

It had long been surmised that micro-organisms had much to do with morbid processes, and that this relationship was that of cause and effect. It was known, for instance, that suppuration, surgical fever, erysipelas, hospital gangrene, and puerperal fever were associated with conditions which favored the multiplication of the lower forms of life. What relationship the micro-organisms bore to the various affections was not known. Least of all were the specific micro-organisms which were the causes of particular specific morbid processes known. I have said that it was surmised; but there was groping about, a difference of opinion, a maximum of theory, a minimum of fact. It is true that in relapsing fever the spirillum had been found, and that none had been found in any other disease. Moreover, it is true that monkeys had been inoculated and the disease reproduced in them. It is true the bacillus of anthrax had been seen in the blood, in the early "sixties." It is true that the great genius Pasteur had prosecuted studies of bacteria in animal and vegetable pathology to most brilliant and practical conclusions. Nevertheless, there was confusion and doubt; scientists were not satisfied with the demonstrations which undertook to prove the causal relationship of micro-organisms to disease.

Laws to Establish Causal Relationship. Through the genius of Robert Koch theories and objections were set at naught. The scientific world was fully prepared by the labors of early investi-

gators to accept Koch's conclusions. They were based upon an array of well-formulated facts, which anyone could prove for himself. Koch's laws were, in substance, that in order to assert that a specific micro-organism is productive of disease we must demonstrate, first, its constant presence in the fluids or tissues of the individual subject to that disease; second, its absence from all other diseases; third, its isolation, growth, and repeated cultivation on proper culture media; fourth, its power of reproducing the disease after inoculation in susceptible animals. The experimental circle was then repeated. In this manner the causal relationship of micro-organisms to special diseases had been proven by the distinguished investigator in the case of anthrax, tuberculosis, and other affections. Unfortunately there has been limitation to the researches, because of the difficulty, among others, of finding animals that are susceptible to inoculation with some of the micro-organisms capable of producing disease in man.

Aid to Diagnosis. It is readily seen that when the definite cause of an infectious disease has been isolated and the morphological and biological properties of the causal micro-organism studied, the clinician has acquired a valuable aid to diagnosis. Indeed, in such affections, diagnosis has become an absolute certainty.

Method of Research. The diagnosis to be complete must include, (1) the finding of the specific micro-organism in the blood or tissues of the subject or in the pathological secretions or excretions; (2) the isolation and cultivation of the micro-organism; (3) the inoculation and the reproduction thereby of the disease in animals. In many affections the morphological properties of the micro-organism are such that the finding of it is sufficient to establish a diagnosis. On the other hand, in some affections, the absence, or rather failure of detection, of the micro-organism in the fluids or discharges is not proof that the disease is not present in the suspected individual, in whom symptoms and lesions point to a specific micro-organism. The affection tuberculosis well illustrates the propositions in the last two sentences. If the bacillus is found in the sputum of a suspected case the diagnosis is established definitely, and no further procedures for diagnostic purposes are necessary. In other clinical forms, as tuberculous pleurisy, or empyema, or glandular or joint tuberculosis, the micro-organisms are few and difficult to find. Cultures, or more conclusive still, inoculations, must be resorted to, often, before a final conclusion can be arrived at. It is possible that spores alone exist—morphological elements difficult to detect by staining and microscopical methods, but which may rapidly multiply under favorable culture or inoculation conditions. Again, micro-organisms have been found in certain affections, and although thus far their causal relationship to the latter has not been fully proven, nevertheless their constant occurrence in the special affection and in that one alone, renders their presence of high diagnostic value. Thus the amœba of dysentery and the plasmodium malariæ of Laveran are diagnostic of their respective affections.

Essential Knowledge. For diagnostic purposes, bacteriological research must be conducted in accordance with the methods of bacteriology. Such researches are possible at this time, because of, 1, the high degree of development and mode of use of optical apparatus, including oil-immersion lenses, Abbe's condenser, and diaphragms; 2, the development by Weigert of the effect of aniline dyes on protoplasm, and the property of micro-organisms of taking different stainings; 3, of the principles of sterilization by heat, by which foreign micro-organisms are excluded; 4, of the use of solid culture media, and the plate method of obtaining pure cultures suggested by Koch.

Bacteria. Varieties. Bacteria are of two classes. One class obtain subsistence from dead organic matter, breaking it up into such simpler forms as carbon dioxide, ammonia, etc. They act to some degree as scavengers, and are beneficial rather than harmful. Such are called *saprophytes.* The second class live at the expense of higher forms of life, and at the same time produce very poisonous substances. They are called *parasites*, and are or are not essentially harmful. We are concerned with the harmful varieties. They imply the presence of a host in which they develop. They may enter the blood. Diseases to which they give rise are known as *infectious diseases.* The process they set up may be local, as in gonorrhœa or certain skin affections, or general, as in typhoid fever, syphilis, or tuberculosis. In some instances it is first local and then becomes general, as in tuberculosis. Their clinical manifestation is seen in the infectious diseases. Sometimes certain bacteria of one class may acquire the power of living like those of the other class, and are then called *facultative saprophytes* or *parasites.* They develop in cavities of the body. They may enter the blood. They produce in certain cases particularly poisonous substances which enter the circulation and cause an intoxication, to which the term *sapræmia* or *toxæmia* is applied. Parasites and facultative parasites include those bacteria that are productive of disease, and are therefore known as *pathogenic bacteria.* All bacteria require certain conditions and certain materials for their development. All require carbon, nitrogen, and water, and a certain temperature, which varies in each case. Some require oxygen and are called *aërobic;* others cannot grow in the presence of oxygen, and are called *anaërobic.* Others grow either with or without oxygen. These are called *facultative anaërobic.*

Morphology and Biological Characteristics. To determine the micro-organism which may be the cause of the disease under examination the student must be familiar with the morphology and the biological properties of the various forms. By these means a distinction between them is possible, and a bacteriological diagnosis made. *The morphology.* The shape, the size, the mode of reproduction and grouping are to be studied. Bacteria or fungi are divided morphologically into *micrococci* or spherical bacteria, *bacilli* or rod-shaped bacteria, and *spirilla* or twisted forms. Bacteria procreate by simple fission, and are therefore known as *fission fungi* or schizomycetes. Some forms also produce spores. *The biological properties* include motility, color, the growth on various culture media, and under various temperatures, and the products

of vital activity. The growth on various culture media will be considered under each pathogenic bacterium which it is the province of this work to discuss. On the character and extent of this growth, its color and other properties, data are collected by which the various microorganisms are distinguished. Some properties which do not belong to pathogenic bacteria will not be considered, as the production of phosphoresence, the production of marsh gas, hydro-sulphuric acid, viscous fermentation, and the fermentation of urea.

By the above we can sufficiently identify the pathogenic bacteria for our present purpose.

Micrococci. MORPHOLOGY. To this group belong the spherical bacteria. Each coccus is of equal diameter in all directions. They vary in size from 0.1 μ to 1 or 2 μ. A micromillimetre (μ) is one twenty-five thousandth of an inch. The various micrococci resemble each other so much in form and size that they cannot be distinguished by their microscopic appearances. To distinguish them, dependence must be placed on the color and character of their growth in various culture media, pathogenic power, and other biological differences. The mode of grouping after fission or reproduction is an important characteristic by which varieties are differentiated. Just before dividing, they are not exactly spherical, but short or long oval. After division, the *staphylococci* (for they divide indefinitely) are solitary or in pairs, or occasionally in groups of four, or in clusters roughly likened to a bunch of grapes. The organism is a *diplococcus* when associated in pairs. Sometimes two or four are included in a capsule. *Zoöglœæ* are groups of cocci held together by a transparent glutinous substance. *Streptococci* are characterized by grouping in chains, known as *chaplets*, or *torula* chains, because division takes place in one direction only. When division takes place in two directions, groups of four, or tetrads, are formed ; and when in three directions, groups or packets of eight are formed, of which the *sarcinæ* are the most familiar examples.

BIOLOGICAL CHARACTERISTICS. Micrococci are not *motile* and do not form spores. *Products of vital activity.* The distinction of the various forms of bacteria is also made by noting the difference in the products of vital activity. Of these, *pigment production* is one of the most apparent. The staphylococcus pyogenes aureus and citreus are chromogenic or pigment-producing bacteria. The *liquefaction of gelatin*, when cultures are made, is a biological characteristic which points to the diagnosis of the various species. Some pathogenic as well as non-pathogenic germs thus act toward the nutrient medium ; others of both classes do not affect it. A peptonizing ferment is formed during the growth of the cells, which acts upon and dissolves the gelatin. The amount, degree, and form of liquefaction serve to distinguish various species. The staphylococcus pyogenes aureus and albus (as well as others) are liquefying micrococci. *Production of acids.* In the growths of bacteria, many produce an acid—lactic acid, acetic acid, butyric acid—which gives an acid reaction to the culture media. This may be seen if a neutral litmus solution has been added to the gelatin. The pink color produced indicates the presence of an acid. Culture media, it must be remembered,

are alkaline or neutral. The pathogenic micrococci which produce an acid are the staphylococci of pus—lactic acid.

Putrefactive fermentation is set up by bacilli and not micrococci. Other products of vital activity need not concern us, as they are produced by non-pathogenic forms.

Toxic ptomaïnes and *toxalbumins* are products of many forms of pathogenic bacteria, and are the cause of the symptoms of the infective diseases in many instances; thus in diphtheria, the local infective inflammation is due to the bacillus; the general symptoms are due to the toxalbumin. The isolation and detection of the toxalbumins are not sufficiently easy to warrant such mode of investigation for diagnostic purposes. Often the results of inoculation, by which the lethal effect is produced, aid in the diagnosis of the suspected ailment.

The Bacilli. Morphology. The bacilli or rod-shaped bacteria differ widely in form, in size, and in modes of grouping after fission. *Form* and *size*. The longitudinal diameter is greater than the transverse, and the forms vary from short oval or slender rods to long filaments; sometimes short rods and long filaments are seen in pure cultures of the same bacillus, as in the typhoid bacillus. The transverse diameter does not vary, as a rule. The form of the extremities of the rods must be observed. They may be square, slightly rounded, round, oval, or lance or spindle shaped. *Reproduction* and *grouping*. Fission or reproduction takes place by binary division, transverse to the longitudinal axis. They group in long chains, or are solitary, or united in pairs. They may be surrounded by a capsule or collect in zoöglœa masses.

Spores. When conditions unfavorable to continuous multiplication by transverse division arise, certain bacilli possess the property of entering into a permanent or resting stage. In this case there develops within the body of the bacillus an oval, egg-shaped structure—an endogenous spore. The spore represents the inactive stage, and lies dormant until circumstances favorable to growth reappear, when it develops into a bacillus identical with that from which it was formed. Spores do not develop into spores, but into bacilli. The spores retain their vitality for months or years, and resist desiccation. They are spherical or oval, and highly reproductive. They are formed by condensation of protoplasm at the centre or at one end of the bacillus, where they are retained in a linear position until set free. Some bacilli grow into long filaments during spore formation; others change their shape, swelling at the centre, becoming spindle or club shaped, according to the location of the spore within it. Many bacilli do not change their shape at this stage. The spores are free or collected in masses with the bacilli as well as located in the parent bacillus.

Motility. The bacilli are often actively motile, because of the presence of flagella. The movement is one of progression in different directions. It may be slow and deliberate, in a to-and-fro motion, or serpentine, or a quick, darting forward motion.

Biological Characters. *Products of vital activity*. They may be ascertained in the same manner as in the study of micrococci. *Pigment*

production is seen in cultures of the bacillus pyocyaneus or bacillus of green pus, of which there are several varieties producing various shades of blue or fluorescent green. *Liquefaction of gelatin.* This is produced by the bacillus anthracis and the bacillus pyocyaneus. *Production of acids.* The bacillus coli communis produces lactic acid. *Putrefactive fermentation.* The latter bacillus sets up fermentation.

The Spirilla. MORPHOLOGY. They are seen in the form of curved rods or spiral filaments. The shorter ones are curved, the longer are spiral, like a corkscrew. The curved filaments may be short and rigid, or long and flexible.

Reproduction. They reproduce by binary division (fission).

BIOLOGICAL CHARACTERS. *Motility.* They are motile; the movement is rotary, as well as progressive in the direction of the long axis of the filament. The presence of flagella is determined by Löffler's method. They are single at the ends of rods, or several are seen at one extremity, or they are around the entire periphery. *Pigment production.* Pathogenic spirilla do not produce pigment. *Liquefaction of gelatin.* The spirillum of cholera Asiatica (comma bacillus), and the spirillum of cholera nostras (Finkler and Prior) each liquefy gelatin in a peculiar manner.

Methods of Research. Having learned the morphological and biological characters of the various forms of pathogenic bacteria, the student is prepared to render such knowledge useful for diagnostic purposes. I have said that methods of bacteriological research must be employed; the following account is to embrace the steps that should be taken to ascertain the presence of a micro-organism in the blood, the secretions or excretions, the fluids of cavities or cysts (exudations, transudations, and cystic fluids). In a case the character of which is unknown, and in which there is no distinctive pathological discharge or reproduction, all fluids of the body must be examined. In other cases, the pathological discharge (pus), or perhaps diseased tissue, must be examined. We derive a clue as to the direction which the examination is to take by the nature of the symptoms. In cases of pulmonary disease, the sputum; of faucial disease, the membrane, pus, or other secretions from the fauces; in intestinal disease, the discharge from the bowels, and in genito-urinary disease, the urine. It must not be forgotten that in many, even highly fatal diseases, the blood is not invaded by micro-organisms. Death is due to the development of toxic substances. Hence, as in cholera or diphtheria, the presence of the micro-organism is not sought for in the blood, but in the specific excretion or exudation.

The *method of procedure* is: 1. Microscopical examination of a minute particle of the stained and unstained blood or the morbid secretion or excretion. 2. Cultivation of the micro-organisms on plates. 3. Inoculation of animals with pure cultures of the suspicious organism or organisms.

THE APPARATUS. The *apparatus* necessary to the simplest bacteriological research is as follows: Sterilizers, incubator, glass flasks, covered

dishes, test-tubes and plates, platinum needles fixed in glass handles, cotton, materials for culture media, microscope, with slides and cover-glasses, and in addition to lenses of lower powers, a $\frac{1}{12}$ oil-immersion lens, and finally the various stains used.

Preparation of apparatus. Boil all glassware for half an hour in a solution of common soda (4 to 6 per cent.), then scrub thoroughly, rinse in warm solution of $HgCl_2$ (1 per cent.), and then in pure water, drain with tops down; plug tubes and flasks with raw cotton, fitting firmly and evenly, so that the cotton can hold the weight of the test-tube; sterilize in dry oven. The test-tubes (plugged) are placed in a rack for further use.

The tubes and flasks are best filled with the culture media through a spherical funnel that can be plugged with cotton. Then they are to be sterilized in the steam sterilizer as heretofore described.

The cover-glasses must be thoroughly cleaned by immersion in strong nitric acid for a few hours, then rinsed in water, then in alcohol and ether. They are then kept in alcohol.

Sterilization. It should be understood that the first requisite for the prosecution of these studies is to secure absolute cleanliness and to prevent the invasion of extraneous micro-organisms. The first step is thorough sterilization of all appliances required for work, and of all the media, to destroy previously existing bacteria.

The sterilization is best accomplished with steam where the objects to be sterilized admit of it. With dry heat, a temperature of at least 150° C. must be applied for at least an hour, and of course can only be used for glassware and metal instruments. All media (see page 154), whether solid or fluid, are sterilized by steam. Media which cannot withstand long exposure to the necessary heat are sterilized by the intermittent application of steam. The reason that this is effective is that fully-developed bacteria are destroyed at a much lower temperature and with shorter exposure than are the spores. One application kills the developed bacteria, then the material is kept for a time in an incubator, spores develop into bacteria and are easily killed by a second application. By repeating this process from three to five times the substance is effectually sterilized. If the exposure is made longer a much lower degree of heat may be used, so that in the case of blood-serum it may be sterilized without coagulating the albumin. Usually an exposure of fifteen minutes to steam on each of three successive days is used for stable media, and an exposure of an hour on six successive days to a temperature of 70° C. for more delicate media, as blood-serum. In the intervals the materials must be kept at a temperature of 25° to 30° C. A single application of steam under pressure is often used, but only very stable materials can be subjected to this without damage.

The ordinary "Arnold steam sterilizer" is as good as any. The dry sterilizer is merely a metal box with copper bottom and ventilating holes. It is well to have an asbestos casing.

Metallic articles, as forceps, platinum probes, etc., are best sterilized in the flame of a Bunsen burner.

Collection of Material. A definite careful method must be observed when the pathological product is removed from the patient, or collected for investigation (see Chapter V.—Exploratory Puncture). Pus and fluids should be placed in sterilized glass bottles or tubes, care having been taken that instruments for the removal of the fluid were previously sterilized. Exposure to air should be as brief as possible. The fluids should not be contaminated with blood or antiseptic fluids used for flushing or other surgical procedure. If an abscess is opened or purulent peritonitis cut down upon, for instance, tube inoculations can be made at the bedside. The previously sterilized platinum point should be kept before use in a test-tube, closed with sterilized cotton. It is dipped into the pus, which should be free from the blood of the incision, but before it flows over the skin. It is at once applied on the media of the test-tube. Sputum should be collected in a previously sterilized bottle, or one thoroughly cleansed by boiling. The bottle should have a wide mouth. Care must be taken to secure sputum from the lungs, and not the secretion from the mouth and fauces. Purulent portions, rather than mucoid, are to be sent for examination. Blood should be examined at the bedside microscopically, and cultures made at the same time. Cover-slip preparations may be made at the bedside for future staining. Intestinal discharges may be collected in sterilized glass jars and examined as soon as practicable. It may be necessary to keep the discharge at the temperature of the body. (See Fæces—amœba dysenterica).

To secure blood for microscopical study, the finger must be thoroughly cleansed with alcohol and puncture made with a sterilized lancet or needle. After the blood flows a few seconds it is removed and the cover-slip, previously cleansed in nitric acid solution, is gently pressed upon the second overflow. Another cover is placed over the blood-stained surface of the first slip, the two rubbed together and separated by sliding them apart. Sternberg prefers to spread the blood, which was collected at the edge of the cover-slip, by drawing a polished glass slide, held at an acute angle, over the cover-slip. In either case this thin film of blood is allowed to dry, and can be examined later. Sternberg mounts the blood on a glass slide at once.

Microscopical Examination. The blood and fluids, stained and unstained, and colonies of the preliminary and pure cultures are examined. The methods for each as to technique are about the same. The cover-slips that are stained must be examined with the oil-immersion objective, and the diaphragm of the sub-stage condensing apparatus (Abbe's) open. When not stained the diaphragm must be closed.

The *blood* may be examined without staining. The bacillus of anthrax and the spirillum of relapsing fever may be thus detected. Basic aniline dyes are used to stain the cover-slip preparation or the method of Löffler or Gram employed.

The *secretions* in general are examined by the same method. By Günther's method the spirillum of relapsing fever is detected in the blood. Examination of the blood, and the *sputum* for tubercle bacilli and other micro-organisms, will be described in the section on Sputum.

The examination of the nasal and buccal secretions is described in the appropriate chapter. Gram's and Günther's methods are of value. Search for the bacteria in the alimentary tract (see Vomit and Fæces) must be made in accordance with methods described in those sections and by the methods of staining hereafter described. The *urine* is studied with the Gram and the Friedländer method. The study of pus will be described later.

Examination of Colonies. Just here may be stated the methods employed for the study of the morphology of the colonies secured by plate and other means of cultivation. The same process applies to the examination of pus and pathological fluids.

Cover-glass preparations are made as follows: On the cover-glass place a small drop of distilled water. With a platinum needle take up the smallest possible quantity of the colony to be examined, mix it with the drop and spread over the surface of glass. Dry under cover or by holding *with fingers* over a flame, the layer of bacteria being away from the flame. When dry, pass it with forceps three times through the gas or alcohol flame to "fix" the albumin. It is then ready for staining.

Methods of Staining. Many have been devised, but those of clinical value are the following:

1. Aqueous solutions of basic anilines.
2. Löffler's alkaline methyl-blue.
3. Koch-Ehrlich's aniline water solutions.
4. Ziehl's carbol-fuchsin.
5. Löffler's method of staining flagella.
6. Gram's method.
7. Friedländer's method.
8. Günther's method.

1. Basic anilines. Aqueous solutions of the basic aniline colors—fuchsin, gentian-violet, and methyl-blue—are used in such strength that they can be seen through clearly in an ordinary test-tube. They may be kept on hand in bottles with pipettes, or made from concentrated alcoholic solutions as needed. They are used by simply dropping a few drops on the cover-glass preparation, which is held with the forceps, allowing it to remain about thirty seconds, and carefully washing off in water. It is placed on a slide, *bacteria down*, and the excess of water removed with blotting paper.

2. Löffler's alkaline methyl-blue solution. Certain bacteria take a stain more readily when an alkali has been added. The formula is as follows:

Concentrated alcoholic solution methyl-blue	30 c.c.
Caustic potash, 1 : 10,000	100 "

It is used in the same way as the simple solutions.

3. Koch-Ehrlich aniline-water solutions. Add to 100 c.c. of distilled water, aniline oil, drop by drop, thoroughly shaking after each drop, until it becomes opaque. Then filter. Add 10 c.c. absolute alcohol and 11 c.c. of a concentrated alcoholic solution of either fuchsin, methyl-blue, or gentian-violet.

4. Ziehl's carbol-fuchsin solution.

Distilled water	100 c.c.
Carbolic acid	5 gm.
Alcohol	10 c.c.
Fuchsin	1 gm.

The use of these various stains will be described in the description of the different bacteria.

5. Löffler's solution for flagella.

Tannic acid, 20 per cent.	10 c.c.
Cold saturated sol. ferric phosphate	5 "
Saturated solution fuchsin	1 "

A few drops of this are placed on the cover-glass containing the blood or pus and heated until it begins to steam, and then washed off in water. The preparation is then stained with aniline water fuchsin. Different bacteria require different reactions, and so a few drops of an acid or alkaline solution are added as the case requires.

6. Gram's method consists in staining with a Koch-Ehrlich solution for twenty to thirty minutes, and then decolorizing in

Iodine	1 gm.
Potassium iodide	2 "
Distilled water	300 c.c.

After remaining in this for five minutes, preparations are rinsed in alcohol, and the process repeated until the violet color has disappeared.

For Günther's and Friedländer's methods, see Sputum.

To detect *spores* of bacilli double staining may be employed. The preparation is first stained in a hot Ziehl-Neelsen fuchsin solution, then decolorized with nitric acid. When stained again with methylene-blue, the spores appear red, the bacilli blue.

The "hanging drop." By the examination of colonies in the *hanging drop*, we learn of the movement of the micro-organism. Place a drop of salt solution on a cover-slip, and add a tiny portion of colony on platinum wire; place the slip, drop down, on a glass side in the centre of which is a depression or hollow. Fix the slip by applying a thin layer of vaselin around the margin of the depression. Care must be taken in focussing that the lens does not break the glass, readily done because of the transparency. The bacteria are seen in motion; on account of the motion their position is constantly altered. This motion must not be mistaken for the Brownian movement of suspended particles, which is vibratory from molecular tremor.

Cultivation of Micro-organisms. The object to be obtained is to isolate the pathogenic organism from all other organisms, and to exclude organisms that may be introduced from without by unclean instruments or other means. *Pure* cultures of the fungus are thus obtained.

CULTURE MEDIA. Experience has taught us that various forms of bacteria require different pabulum, and that various nutrients are required for the isolation of different micro-organisms. As to the bacteria hereafter noted, we are familiar with the proper soil for their growth. The media used for bacteria of clinical importance are: freshly steamed potato, gelatin, bouillon, agar-agar, milk, and blood-serum. They are

prepared or mixed in various ways, and other things may be added, as a solution of litmus, to determine the reaction of the bacterial products.

Bouillon. Lean beef, 500 gm., soaked in one litre of water for twenty-four hours in ice-chest; strain through a coarse towel and press until a litre of fluid is obtained. Add 10 gm. of dried peptone and 5 gm. salt. Then neutralize with a normal solution (4 per cent.) of caustic soda. Boil till albumin is coagulated, filter, and sterilize.

Nutrient Gelatin. Make bouillon as above (except neutralizing) and add 10 to 12 per cent. of gelatin, and neutralize after dissolving it by heat. Filter.

If not perfectly transparent, clarify by heating to 60 to 70° C., add whites of two eggs beaten up with 50 c.c. water; mix thoroughly and boil until albumin coagulates; then filter. Sterilize, and keep in flasks or tubes.

Nutrient Agar. Prepare bouillon complete; add finely chopped agar, 1 to 1.5 per cent. Place in a porcelain-lined iron vessel, mark level of fluid, add 250 c.c. of water and boil slowly, with occasional stirring for three or four hours. Keep the fluid up to mark by adding water. Take the vessel from the fire and set it in cold water. Stir until cooled to 68° to 70° C.; add the whites of two eggs beaten up in 50 c.c. water. Mix carefully and boil for half an hour, keeping fluid up to the level. Filter.

Sometimes 5 to 7 per cent. of glycerin is added.

Potatoes. Select old potatoes; scrub under water faucet with stiff brush; cut out eyes and defects. Then place in 1 : 1000 $HgCl_2$ for twenty minutes. Then place in steam sterilizer and steam forty-five minutes. Leave them in and steam fifteen or twenty minutes each day for three days. Cut with knife sterilized in flame and lay with cut surface upward in a sterilized covered dish.

Another way of preparing potato is to cut cylinders with a cork borer of such size as to fit loosely in a test-tube. A slanting surface is then cut from the junction of the first and second thirds of the cylinder to the diagonally opposite edge. These are left in running water over night, then placed in test-tubes with a cotton plug and steamed for forty-five minutes. On the second and third days they are steamed fifteen to twenty minutes.

Milk. It should be sterilized and peptonized. It is a good soil for the tubercle bacillus. (Abbott.)

Blood-serum. This is difficult to prepare. Glass jars with tight covers must be carefully sterilized and dried. The animal (at slaughter-house) is drawn up by hind legs and the throat cut by one stroke, and then the blood caught in the jars. The covers are fastened loosely and the jars allowed to stand about fifteen minutes until clotting has begun. Then a sterilized rod is passed around the edge of the clot to break all adhesions to the sides of the vessel. The covers are then replaced and the jars placed in an ice-chest for twenty-four to forty-eight hours. Then draw off the serum with a sterilized pipette into tall sterilized cylinders, and plug them with cotton. Then place again in ice-chest for twenty-four hours to settle. Then draw off either into test-tubes (each 8 c.c.) or into flasks. Sterilize by the intermittent method at low

temperatures. If desired the serum can be solidified by exposure to dry heat—78° C.—for two hours. Then the tubes must be sealed with rubber caps to prevent drying.

Löffler's blood-serum mixture.

Neutral meat infusion bouillon (see Bouillon)	1 part.
Grape sugar	1 per cent.
Blood-serum	3 parts.

Tube and Plate Cultures. The plate method was introduced by Koch for the purpose of isolating individual bacteria from a number of them. It may be practised either with gelatin or agar-agar. Three tubes previously filled with the culture media are taken and liquefied by warming in a water bath, then cooled to the lowest point at which the medium remains fluid. One of the tubes is then taken and held in the left hand. A sterilized looped platinum wire inserted in a glass handle is taken in the other hand, passed through a flame and cooled for a few seconds. With this a bit of the material to be examined is taken up, the cotton plug is removed from the tube with the free fingers and the wire inserted into the medium. By rolling the tube it is thoroughly mixed. Then in the same way a second tube is inoculated from the first, and a third from the second. Plates have been previously sterilized and placed in covered dishes also carefully sterilized. The plates are levelled and the contents of tubes poured upon their surface. Then they are cooled over ice-water until the medium becomes solid, when they are placed in a proper temperature for development. In this way the bacteria are sufficiently diluted to form distinct colonies from which pure cultures may be obtained.

A convenient modification of the method is the use of *Petri's plates*, which are flat, round dishes with covers, the bottom of the dish serving as the plate.

Another modification (*Esmarch's tubes*) is the use of tubes with a small quantity (5 c.c.) of the medium. By rolling the tube in the fingers the sides are coated with the media. They are then rolled on ice, so that the medium solidifies in a thin layer about its walls.

Smear and Stab Cultures. When the bacteria have been isolated by one of these methods, pure smear or stab cultures must be made from them. A tube of the proper culture medium is taken in the left hand, a bit of a pure colony taken up on a sterilized straight platinum needle, the cotton plug removed as above, and the needle thrust straight into the medium for a stab culture, or rubbed over a slanting surface of media for a smear culture. The plug is immediately inserted and the tubes transferred to the incubator.

When pure cultures have been obtained the species are recognized by their *mode of growth* and behavior in different culture media, the *reaction* produced by their growth, and their appearance under the microscope when stained and unstained.

When nutrient media are inoculated they must be placed in favorable conditions as to *temperature*. This will be detailed when each microorganism is discussed, as a number of pathogenic bacteria require a definite and continuous temperature.

The primary inoculation will often yield numerous colonies the nature of the organism of which must be determined by its morphology and biological characteristics. Frequently each colony must be again cultivated before complete isolation of the specific bacterium is produced.

Inoculation of Animals. Another method of determining the pathogenic character of morbid material, as sputum, pus, or exudation, is by inoculating animals with a pure culture. This is done either by feeding, by subcutaneous injection, or by injection into the circulation, with antiseptic precautions.

As animals are subject to only a few of the microbic diseases, many experiments must often be made before a susceptible animal is found, and no conclusion can be reached as to the pathological power of a micro-organism until this point has been determined. The clinical course of the artificial disease must be observed to fulfil the diagnosis.

Examination of the animal is made as soon as possible after death. The autopsy is made with antiseptic precautions. After the skin is removed only sterilized instruments are to be used. The macroscopical appearances and mode and progress of infection are noted to aid in the diagnosis. When the organs are exposed, material for cultures is first obtained by inserting a platinum needle through a small puncture in the capsule. Afterward cover-glasses may be prepared for immediate examination. Blood is taken from one of the cavities of the heart. After the autopsy all remains are to be burned, and all instruments carefully sterilized.

Special Bacteriological Diagnosis. In the preceding section the general methods were described by which the micro-organisms were searched for. As they are found in different fluids or secretions of the body, a discussion of the individual forms the detection of which implies an absolute diagnosis, will be considered in different sections which treat of the special diseases, or the special fluid in which the organism is most frequently found. In the subsequent chapter the method of examining pus will be detailed. In that section an account of the pyogenic bacteria (the morphology and bacteriological characteristics), staphylococcus and streptococcus, will be found. The bacillus of syphilis, the gonococcus, the fungus of actinomycosis, the bacillus of glanders, of anthrax, of leprosy, and of tetanus, will be given. An account of the micro-organism of pneumonia and that of tuberculosis will be found in the section on sputum, of diphtheria in the section on the pharynx, of cholera in the section on intestinal diseases (fæces), and of typhoid fever in its appropriate section. In the section on disease of the blood, and in the special articles the spirillum of relapsing fever and the protozoa of malaria will be discussed.

The following points must be investigated in order to determine the specific nature of the micro-organism which is supposed to be the productive agency of the disease in question, viz.: The *form*—micrococci, bacilli, spirilla, polymorphous; *relation to oxygen*—aërobic, facultative

anaërobic, strict anaërobic; *growth in nutrient gelatin*—liquefy, do not liquefy, do not grow at "room temperature;" *growth on potato; growth on milk*—coagulate milk, do not coagulate, etc.; *color of growth*—chromogenic, non-chromogenic; *spore formation; movement; pathogenic power.*

NOTE.—For further information concerning technique the student must refer to the work of Abbott on the "Principles of Bacteriology," and to Sternberg's "Manual of Bacteriology" for an exhaustive account of the technique, and the morphology and bacteriological characteristics of all bacteria, pathogenic and non-pathogenic. The text-books of Hueppe, "Die Methoden der Bakterien-Forschung," 1886; Baumgarten, "Lehrbuch der pathologischen Mykologie," 1890; Fluegge, "Die Micro-organismen," 1886; and Cornil and Babes, "Les Bactéries," 1890, are profitable for the further prosecution of studies.

CHAPTER V.

THE EXAMINATION OF EXUDATIONS, TRANSUDATIONS, AND CYSTIC FLUIDS.

Exploratory puncture or aspiration for diagnosis: Instruments. Preparation of Instruments. Preparation of skin. Point of puncture.—*Exudations* (Pus. Sero-pus. Gangrenous débris. Blood Serum. Chyle): Pus. Blood corpuscles. Bacteria. Protozoa. Vermes. Crystals.—*Chemical examination:* Sero-purulent exudations. Putrid exudations. Hemorrhagic exudations. Serous exudations. Chylous exudations. Pleural effusions. Transudations.—*The contents of cysts:* Hydatid, ovarian, renal, pancreatic.

Exploratory Puncture or Aspiration for Diagnosis.—The presence or absence of fluids in the natural cavities of the body, as the pericardium, the pleura, or the abdomen, or in the gall-bladder, must often be ascertained by means of puncture or aspiration. The fluid is also thus secured to determine its nature. The fluid of tumors or cysts is likewise withdrawn to complete a diagnosis by determining its chemical, microscopical, or bacteriological character. Certain rules of procedure are necessary, and, as they belong in common to the method in whatsoever situation employed, may be considered in this section.

The Instruments. If it is the desire of the observer to determine the presence of fluid, an ordinary grooved needle may be used. If, however, fluid is to be obtained for research, a syringe or aspirator must be used. An ordinary hypodermatic syringe, or the syringe of Pravaz, may be used if the needles are long enough. A special aspirator made for diagnosis by instrument-makers is the best. The needles are sufficiently long, the barrel large enough to hold enough fluid for each method of examination. If the diagnosis is to be followed by treatment by aspiration, the apparatus of Dieulafoy, or any equally perfect apparatus, may be used at once.

Preparation of Instruments. The instruments should be sterilized in a steam sterilizer, or boiled. This does not apply to the needles alone, but every portion of the instrument should be cleansed, because, for instance, the contents of the barrel of the syringe pass through the needle when testing it. After sterilization they should be carried to the patient in sterilized test-tubes plugged with cotton wool. After boiling, the needles should be kept in absolute alcohol, and the syringe in carbolic acid solution, 1:20, twenty minutes before operation.

Preparation of Skin. The skin should be first cleansed with soap and water, then with alcohol, then with a solution of carbolic acid, 1:20, or of the bichloride of mercury, 1:1000. After thorough cleansing, the parts should be kept covered with a towel soaked in bichloride solution until the time of operation. At the time of puncture the sur-

face should be made anæsthetic by ethylene chloride, the rhigolene spray, or by ice and salt. Care must taken, if the patient is aged or poorly nourished, or the skin œdematous, not to freeze the skin too much, on account of the danger of local gangrene.

The Point of Puncture. The points selected for aspiration depend upon the cavity the contents of which are explored, or the situation of the cyst.

The Pleura. To determine the nature of fluid within the pleura it is best to select a point for aspiration in one of the lower interspaces of the chest, because the fluid is more likely to accumulate in this position and because complete aspiration can be performed if necessary. The sixth or seventh interspace in the anterior axillary line, or the eighth or ninth interspaces in the posterior axillary or scapular line may be selected. On the right side, the uppermost interspace of the two should be chosen on account of the position of the liver. If the contents tend to point or break out at any particular spot on the surface of the chest, the puncture may be made in this area.

The Pericardium. For aspiration of the pericardium three points of election have been recommended: first, the usual position of the apex beat, in the fifth interspace inside of the mid-clavicular line; second, the space between the ensiform cartilage and the left seventh cartilage, the point advised by Roberts; third, Rotch has tapped the fifth right interspace a number of times on the cadaver, and thinks that this situation is a proper one on the living subject. The writer has aspirated the pericardium in several instances inside of the normal position of the apex. Care must be taken to insert the needle slowly and with the point directed downward and toward the left axilla when this position is selected.

The Abdomen. It should be remembered at first that no attempts at puncturing the abdomen should be made if pus is suspected, unless preparations have been made to perform laparotomy at once. Indeed, at the hands of modern surgeons this exploratory operation is performed with such little detriment to the patient that on the whole it should be advocated instead of the method of puncture. There are times, however, when the latter must be resorted to. The writer has performed it in a number of instances—always refusing to do so in cases in which pus was probably present in the peritoneal cavity, or in tumors, or in organs connected therewith—without any danger having ever arisen. Explorations of this character are probably more feasible in connection with diseases of the *liver*. It does not appear to be harmful to insert needles into that organ, and valuable information is often gained thereby.

In aspiration of the abdomen, to determine the character of peritoneal contents, the median line should be selected for the puncture. The bladder must be emptied and a point midway between the umbilicus and pubes selected.

Cysts or *tumors* with fluid contents should be punctured over the point which presents externally, at which place it is evidently in closer apposition to the external wall.

The *spleen* has been punctured for therapeutic and diagnostic purposes. If the organ is hard it may be done without danger, but if it is

enlarged and soft as in infectious diseases, such as typhoid fever, it is hardly justifiable to puncture it, because of the danger of subsequent rupture. Risks attend the puncture of other organs, as the kidney. The writer has seen a serious hemorrhage follow such puncture, and of course septic inflammation may arise. Exploratory operation is more suitable for determining its condition.

The Examination of Fluids and Discharges. While the fluids to be considered are obtained by the above-mentioned method, it sometimes happens they can be examined when discharged spontaneously, as in the case of an empyema.

The following general methods apply to the examination, from whichever of the above-mentioned sources the material is obtained. When derived from the natural cavities they are known as exudations or transudations. Fluids are also obtained, however, from cysts, but these do not require different methods of examination.

The naked-eye appearances are first noted; then microscopical examination with and without staining is resorted to. Chemical examination is also required. Often, as in the case of pus or of serous exudation, culture preparations and inoculations must be resorted to.

The Exudations. They may be composed of pus, sero-pus, gangrenous débris, blood, or pure serum, or chyle. When pus, sero-pus, or putrid fluid is withdrawn, it implies absolutely an inflammatory origin. Blood and serum may be associated with inflammation, simple or infectious, but also point to impediments in the general or lymphatic circulation. Blood or bloody serum is thought to be of tuberculous or cancerous origin. Its absence does not imply the absence of either disease. A chylous exudation is usually due to obstruction of the lymph channels.

Pus. Pus ranges in color from gray to greenish-yellow. It is turbid, of high specific gravity, and alkaline. It varies in consistence. When standing after removal it separates into two layers; the upper layer is light yellow and transparent, and the lower opaque. Pus may be mixed with blood, and is then reddish-brown. (See Abscess of the Liver.) When it has undergone decomposition it is thin, green, or brownish-red, of a penetrating odor.

Microscopical Examination: White Corpuscles. If the specimen is fresh the cells exhibit the movements that are common in leucocytes. If a solution of iodine and iodide of potassium is added to them they change to mahogany color. If the pus is old or the cells are dead, they are shrunken and granular. Enormous giant-cells and cells loaded with fat are seen in the pus.

Red Corpuscles. In fresh pus, red corpuscles are also seen along with blood pigment or hæmatoidin crystals.

In addition to the corpuscles, fat globules and particles are seen free. Epithelium is rarely seen. In the pus from the pleural cavity, if cancer is present, the vacuolated epithelial and endothelial cells sometimes seen in cancer may be observed.

Bacteria. Micro-organisms are always detected with the aid of

staining methods. The micro-organisms are usually the determining cause of the suppuration. Suppuration, however, may be caused by chemical substances, although this is at least of rare clinical occurrence. Of the various fungi found the micrococci and bacilli are the most common. Both pathogenic and non-pathogenic varieties are observed. The most common are the *staphylococcus pyogenes aureus*, and *streptococcus pyogenes*. In the pus of empyema the micrococcus lanceolatus, or pneumococcus, is frequently found, particularly in the empyema that occurs secondarily to pneumonia. The bacillus coli communis is found in abscesses about the peritoneum and in purulent peritonitis, the amœba dysenterica in abscess of the liver and secondary abscess of the pleura and lung. It was found in an abscess of the jaw by Flexner. The micrococci are detected by the staining methods. The method of Gram is the most satisfactory.

After a cover-glass has been prepared and placed in Ehrlich-Weigert's solution of gentian-violet and aniline water, it is put into a solution of iodine and iodide of potassium for two or three minutes. A dull red-brown color is produced. It is then rinsed in absolute alcohol for some time. The micro-organisms are stained dark blue. The iodide of potassium solution is: Iodine, 1 part; iodide of potassium, 2 parts; distilled water, 300 parts. By this method the various forms of micro-organisms just indicated are readily brought out.

THE PYOGENIC BACTERIA. 1. STAPHYLOCOCCUS PYOGENES AUREUS.—This micro-organism is found in acute abscesses and boils,

FIG. 15.

Pus with staphylococcus. × 800. (FLÜGGE.)

sometimes also in infectious osteomyelitis and ulcerative endocarditis. It enters the tissues through abrasions or the hair follicles.

Morphology. In cover-glass preparations they appear as small round bodies scattered among the pus-cells, rarely within them, single, in pairs or clusters. They stain readily with the basic aniline dyes. (See Plate I., Fig. 2, B; and Fig. 15.)

Biological properties. It is aërobic, facultative anaërobic, grows in milk, meat infusions, gelatin, or agar at 18° C. Death-point is 56° to 58° C. after ten minutes' exposure. *Growth.* Make plate cultures on agar-agar. After twenty-four hours in the incubator the plate will be studded with yellow or orange-colored colonies, round, moist, and glistening. In a gelatin stab culture, liquefaction occurs in thirty-six

PLATE II.

FIG. 1.

Streptococcus—Erysipelas.

FIG. 2.

Streptococcus Septicus.

FIG. 3.

Staphylococcus.

FIG. 4.

Diphtheria Bacilli.

FIG. 5.

Typhoid Bacilli.

FIG. 6.

Tuberculosis Bacilli.

To face page 163.]

to forty-eight hours along the puncture, forming a funnel. The whole mass gradually liquefies. At the bottom of the funnel the microbes collect as an orange-colored mass. On potato it grows as a brilliant orange-colored, somewhat lobulated layer. The growth gives off an odor of sour paste. (See Plate II., Fig. 3.)

2. Staphylococcus Pyogenes Albus. It is also found in acute abscesses, but less often than the "aureus," and is less virulent.

It is morphologically identical with the "aureus," but develops no pigment. The surface cultures are milk-white, and the mass at the bottom of the liquefying gelatin is white.

3. Staphylococcus Epidermidis Albus closely simulates the staphylococcus pyogenes albus. It is the most common micro-organism on the surface of the body, and is often present in parts of the epidermis too deep for disinfection save by heat. It is supposed to be the usual cause of "stitch abscess."

4. Streptococcus Pyogenes. It is found in acute abscesses, erysipelas, otitis media, puerperal metritis, ulcerative endocarditis, pseudo-diphtheria, scarlatinal angina, and most purulent inflammations of a phlegmonous character.

Fig. 16.

Streptococcus pyogenes in pus. × 800. (Flügge.)

Morphology. Cover-glass preparations show spherical cocci of varying sizes, which form chains of four to twenty elements, the chains often forming tangled masses. It is stained by the basic anilines or by Gram's method. (See Plate I., Fig. 2, B; and Fig. 16.)

Biological properties. Grows in most media at temperature of 16° to 37° C. (best 30° to 37°), but not on potato. It is a facultative anaërobic, and does not liquefy gelatin. On plates it forms a flat transparent disc of about one-half millimetre diameter. In stab cultures it grows all along the puncture and forms a white opaque granular column. The death-point is 52° to 54°, ten minutes exposure. (See Plate II., Fig. 1, and Fig. 2.)

Inoculated, it causes erysipelatous or phlegmonous inflammation.

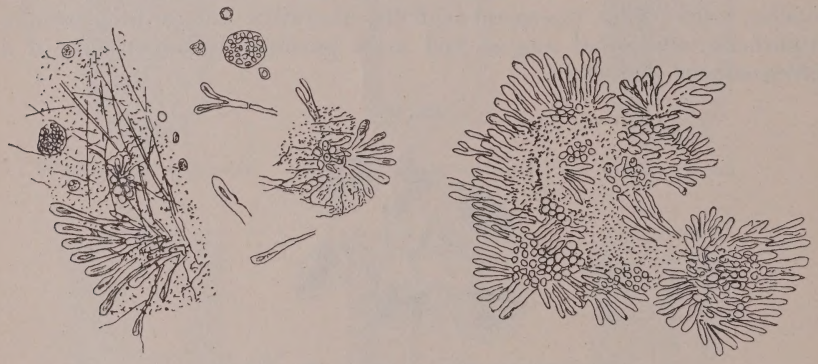
5. The Tubercle Bacillus. This is seen at times in pus removed from phthisical cavities, and the pus of abscesses, particularly about glands. It may be detected by methods of staining adopted in the examination of the sputum. Pus may be of tubercular origin, and the micro-organisms not detected by the usual methods. Its absence, therefore, does not imply the absence of tubercle. Culture methods and inoculation should be resorted to.

6. Bacillus of Syphilis. The pus under these circumstances is usually derived from ulcers or inflammations, or from secretions about the vulva or prepuce.

Lustgarten's method is as follows: After immersion for twenty-four hours at the ordinary temperature in the gentian-violet fluid of Ehrlich-Weigert, the cover-glass preparation is removed and washed for a few minutes with absolute alcohol. It is then placed for ten seconds in a 1 per cent. or 2 per cent. solution of permanganate of potash; a watery solution of pure sulphurous acid is then poured over it, after which it is washed in water. If the preparation still shows color it must be re-immersed for a few seconds in the potash solution and then in the sulphurous acid, and again washed with water.

7. Actinomyces. Israel and Pomfret have given us the greatest amount of information in regard to this parasite. It was discovered by

Fig. 17.



Actinomyces.

Bollinger. It is usually associated with chronic inflammation and the production of pus. The pus is peculiar. It is thin and viscid. Small nodules of a gray or yellow color the size of a poppy seed by the naked eye can be seen when it is spread out on a glass. With a low power these particles are aggregations of spherules, which with a higher power are seen to be arranged in masses radiating from a common centre. Each separate spherule is pear-shaped. They have high refractive power. In the centre of the masses a network of fibres is seen. If the mass is broken up numerous club-shaped forms in the periphery are seen, while at the centre a sort of detritus alone is observed. The micro-organism belongs to the class of fission fungi, and the club-shaped bodies are the degenerated forms. (See Fig. 17.)

Gram's method of staining brings out the threads of the network most distinctly. The centre is made up of a network of minute spherical organisms, with converging constituent threads. The whole is surrounded by a delicate envelope. The pear-shaped bodies may be defined by Weigert's process. Make a solution of 20 c.c. of absolute alcohol, 5 c.c. of concentrated acetic acid, 40 c.c. of distilled water, and a sufficient French extract of litmus to color it ruby red after repeated

filtering. In this solution the cover-glass preparations are allowed to remain for an hour, and then rinsed with alcohol rapidly and placed in a 2 per cent. gentian-violet solution for three minutes. The fluid should be boiled before use, and filtered after cooling. The fungous threads are stained a ruby-red, while the central mass of actinomyces is colorless.

Simple microscopical examination is usually sufficient to determine the nature of the fungus. The recognition is more positive if the peculiar character of the pus is borne in mind in which the nodules are seen, and the club-shaped forms. Pure cultures have been obtained resembling the cultivation of the tubercle bacillus.

8. The Bacillus of Glanders. The pus is usually discharged from the nasal passages. It is detected in dried preparations (see Blood). Löffler's method also enables them to be detected readily. An aniline-water gentian-violet fluid is added to its own bulk of solution of potash 1 : 10,000. The cover-glass is immersed for five minutes in the fluid. It is then removed and placed in a 1 per cent. solution of acetic acid for one minute. The acetic acid should be tinged slightly yellow with tropæolin. The preparation is then bleached by washing in a solution containing two drops of concentrated sulphuric acid and one of a 5 per cent solution of oxalic acid in 10 c.c. of water. The bacillus is also obtained from the pus of an abscess. Its characters are determined by the above methods. It may be cultivated and inoculated in obscure cases. *Growth.* When cultivated, the wet cultivation crop has the appearance of a grayish-white drop. On a potato, at a temperature of 35° C. it grows a thin greasy coating of a brown color. On blood-serum at a low temperature, small scattered transparent drops the color of the serum are seen. It also grows upon glycerin agar-agar and in nutrient milk peptone. Field mice and guinea-pigs are readily infected by inoculation with pure cultures.

9. Bacillus of Anthrax. The pus is derived from the carbuncle in this disease (see Blood). Cultivations may be resorted to, but it can readily be recognized by usual methods of staining. (See Plate I., Fig. 2, *a*.) *Growth.* In the nutrient gelatin medium the bacillus develops in from twenty-four to thirty-six hours. With the glass the scarcely visible minute points are seen to be made up of colonies of an irregular undulating outline, dark in color. After forty-eight hours their shape is more characteristic, and then the cultivation begins to liquefy, stretching over the surface of the plate in wavy stripes. On a sterilized border it forms a whitish gray, slimy patch of uneven surface, scarcely extending over the site of inoculation. On blood-serum the superficial coating of white color is formed. On nutrient gelatin delicately interwoven white threads followed by liquefaction of the gelatin are seen. In drop cultivations in nutrient broths, long shreds develop at regular intervals. Inoculation of the bacillus causes symptoms of splenic fever and the organism is found in the blood.

10. The Bacillus of Leprosy. The micro-organism is found in the nodes, on the skin and mucous membrane. When they break down, abundant thin pus is poured out. The bacilli in large numbers are found. They are in the form of rods 4 to 6 μ, and 1 μ in breadth, and

resemble the bacillus of tubercle. They stain in alkaline fluids, but do not bleach after subsequent exposure to acids. They stain readily (see Sputum). A dry cover-glass preparation must be made and the pus stained with the Ziehl-Neelsen fluid (carbol-fuchsin) and then decolorized in acid and alcohol. It is said that the micro-organism has been inoculated, and also cultivated, although thus far not with diagnostic value.

11. The Bacillus of Tetanus. The bacillus is seen as a delicate, slender rod, with a terminal spore. It stains with aniline dyes and Gram's fluid. Cultivations may be made with the pus. The first cultivations usually contain different fungi. After heating to 80 C. in water bath for half an hour to an hour for several days, gelatin plates, to which 2 per cent. of grape sugar has been added, are inoculated. The plates should be kept, according to Kitasato, in hydrogen atmosphere at 20° to 25° C. If the inoculation is made under the surface of the gelatin, growth begins near the surface. Faint radiating striæ, or thorn-like processes are seen. The development is rapid in agar-agar. After exposure to a temperature of 37° C., after thirty hours the spores make their appearance. On gelatin the colonies are dense at the centre with a more delicate periphery. The preparation becomes fluid and gas is evolved. It is strictly anaërobic.

12. Bacillus of Influenza. (See Sputum.)

13. Micrococcus Lanceolatus. *Pneumococci.* In the pus of empyema, whether from the pleural cavity, or after it has burrowed from other situations, the pneumococcus has been frequently found. It is easily detected by the usual staining methods (for which see Sputum).

14. Bacillus Coli Communis. The bacillus coli communis is found in suppurations about the abdominal cavity (see Fæces).

15. The Gonococcus. It is constantly present in virulent gonorrhœal pus; usually within the pus cell or attached to the surface of epithelial cells. *Morphology.* Micrococci, usually joined in pairs or fours, flattened and separated, when stained, by an unstained intercellular space. Stains easily with anilines—not by Gram's method.

No other cocci are of the same shape, and at the same time within the cells, except one which, however, stains by Gram's method. (See Plate I., Fig. 3, *b*).

Growth. Does not grow readily on media, but can be cultivated on blood-serum; 30°–40° C. is best, and a moist atmosphere is needed. Growth is slow and often fails. Forms a thin, scarcely visible layer, with smooth, shining surface, grayish-yellow by reflected light—is aërobic.

Inoculation into the human urethra produces a typical attack of gonorrhœa.

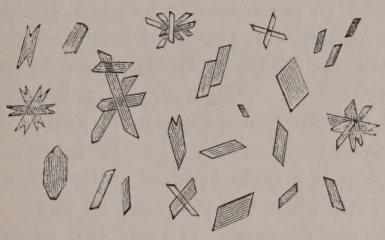
Protozoa in the Pus. Cercomonads have been observed in the pus of an empyema, probably from the lungs. Flexner has found the amœba dysenterica in the pus of an abscess of the jaw. It is found in abscess of the liver and secondary abscess of the lung (see Sputum and Fæces).

Vermes. Filaria have been found in abscess of the liver. In the suppuration of hydatids the pus contains membrane and hooklets.

Crystals. Crystals of *cholesterin* are found in the pus from cold abscesses, suppurating ovarian cysts, and fœtid discharges. They are similar to the crystals described under Sputum.

Hæmatoidin crystals indicate a previous hemorrhage; they are most frequent in suppurating hydatid cysts. (See Fig. 18.) *Fatty needles* are found in old pus and gangrenous exudates. (See Fig. 19.) *Triple phosphates* are frequently seen in pus and are of the same appearance as the phosphates in the urine. The carbonates and phosphates are seen in fœtid pus.

FIG. 18.



Rhombic crystals of Hæmin. (CHARLES)

FIG. 19.

Pus from putrid empyema. (Eye-piece III., obj. 8, A. Reichert). Shrunken leucocytes. Fat crystals. (VON JAKSCH.)

Chemical Examination of the Pus. This does not yield information that is of diagnostic value.

Serum albumin, globulin, and peptone are detected by methods employed in the examination of the urine. Fresh pus contains sugar. After being boiled with an equal weight of sulphate of soda and filtered the filtrate is examined by the reagents used in examination of the urine. In addition to the above, pus contains bile pigments and biliary acids, cholesterin and salts of sodium and the fatty acids in jaundice. Von Jaksch has found acetone in pleural exudations.

SERO-PURULENT EXUDATIONS. They resemble purulent discharges chemically and morphologically. They point to antecedent inflammation.

PUTRID EXUDATIONS. The exudations are brown or brownish-green in color. The odor is penetrating and offensive. They are usually alkaline in reaction. On *microscopical examination*, old leucocytes and crystals of fat, cholesterin, and hæmatoidin are seen; fission fungi of various forms are seen. (See Figs. 18 and 19.)

HEMORRHAGIC EXUDATIONS. Hemorrhagic exudations contain red blood-corpuscles and hæmoglobin in large amount. Fatty endothelial cells are found. Quincke states that when the glycogen reaction is shown, if the fluid is from the pleura, carcinoma is probably present. A positive diagnosis depends upon the discovery of the epithelial cells, which are seen in cases of cancer. Hemorrhagic exudations in the pleura are due most frequently to cancer, to tubercle, or to scurvy.

To determine its exact nature (as to tubercle), inoculation and cultures are sometimes necessary. (See Fig. 15)

Serous Exudations. The fluid is clear and light yellow or straw-colored. On standing, a white fibrinous clot is deposited. On *microscopical examination*, red blood-corpuscles, leucocytes, fatty globules and endothelial cells are found. They may be bunched in groups or scattered about. The micro-organisms, if present, are detected with difficulty. If ulcerating tuberculosis of the pleura is present, the bacillus may be found. Tuberculous pleurisy may exist without ulceration, and hence the fluid is clear of the bacillus. Cholesterin crystals are found in old serum. On *chemical examination* the fluid contains serum albumin and globulin; peptone is absent; sugar in small amounts, and acetone in pleural exudations.

The specific gravity of the fluid is above 1018.

Chylous Exudation. Sometimes in peritoneal exudation, particularly if the patient has been upon a milk diet, the fluid contains fatty matter which gives it a milky appearance. The same character of fluid is seen in obstruction of the thoracic duct. True chyle is found in fluids of low specific gravity. Such an effusion is rich in fat and is due to leakage of lymphatics into the peritoneal cavity. It is known as a chylous effusion. Chyliform effusion is a term applied to the effusion first mentioned in this section. The fluid has the property of chyle.

Special Effusions. *Effusion in the Pleura.* It is of the greatest importance to distinguish the various forms. Bacteriological examination is often necessary. In purulent exudation, if micro-organisms are absent (staphylococcus and streptococcus) it is probably tuberculous; sero-fibrinous exudations are usually free from fungi. When the *micrococcus lanceolatus* is found it is of favorable prognostic omen.

To distinguish the *effusion of inflammation* from that of *transudation* (obstruction) the specific gravity is of service. In the inflammatory effusions the specific gravity is high; the latter also contain a large amount of fibrin.

Transudations. This class of fluids is serous, bloody, or chylous. The specific gravity is lower than in inflammatory effusion. They are light in color; usually alkaline. On microscopical examination but little is found. In pleuritic effusions there may be considerable endothelium which, if with blood, may be due to carcinoma. Serum contains albumin and sugar, the former in great excess. Peptone is always absent. The fluid coagulates with difficulty on boiling.

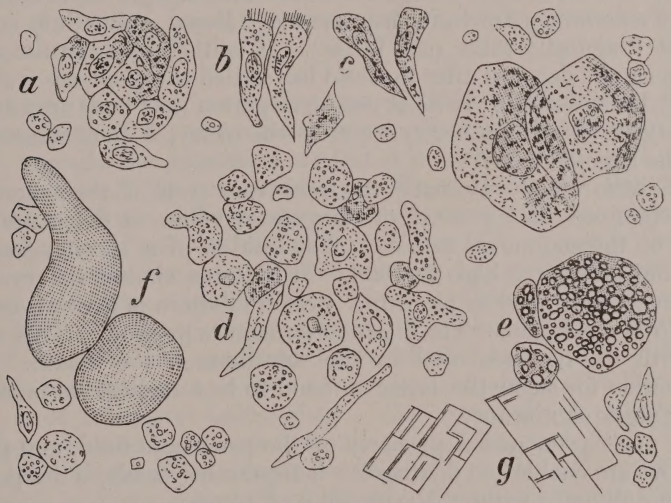
Contents of Cysts. In the aspiration of the abdomen, and sometimes of the pleura, cysts are evacuated, the nature of which is often determined by an examination of the fluid. It is within the province of this work to discuss hydatid cysts, pancreatic cysts, and the cystic kidney. As tumors of the ovary so frequently resemble tumors in other situations, it is well to discuss in this section the nature of the fluid withdrawn.

Hydatid Cyst. The fluid of hydatid cyst is clear, alkaline, and of specific gravity of 1010. It contains chloride of sodium in excess,

grape sugar in small amount, and very little, if any, albumin. On microscopical examination hooklets, as in the sputum from the same cause, are found, and portions of membrane. The membrane is recognized by its peculiar transverse striation and the granular appearance of its inner surface. The heads or scolices are sometimes found. Two circles of hooklets and four disks on the anterior aspect cross the head, which is separated from the hinder part by an annular constriction (see Sputum and Fæces). If suppuration has taken place the original nature of the cyst cannot be made out unless hooklets are found. On standing in a conical glass vessel the bodies may be found in the sediment.

Ovarian Cysts. The fluid from an ovarian cyst is of high specific gravity, 1026, of alkaline reaction, contains but a small amount of albu-

FIG. 20.



Contents of an ovarian cyst. (Eye-piece III. obj. 8, A. Reichert.) *a*, squamous epithelial cells; *b*, ciliated epithelial cells; *c*, columnar epithelial cells; *d*, various forms of epithelial cells; *e*, fatty squamous epithelial cells; *f*, colloid bodies; *g*, cholesterin crystals. (VON JAKSCH.)

min, and does not coagulate. On microscopical examination various forms of epithelial cells are seen, colloid bodies, and cholesterin crystals. If hemorrhage has taken place in the cyst the color of the fluid is correspondingly changed, and in addition to squamous, columnar and ciliated epithelium, some in the stage of fatty degeneration, and red and white blood-corpuscles are seen. In colloid cysts the usual concretions are found. (See Fig. 20.)

In *dermoid* cysts, in addition to the above, squamous epithelium, hairs, and fatty, hæmatoidin, and cholesterin crystals are detected. *Ovarian fluid* contains albumin and methæmoglobin, or paralbumin. The latter is detected by mixing a portion with three times its bulk of alcohol. It is then allowed to stand for twenty-four hours, when it is filtered. The precipitate is removed and suspended in water. After filtering, the filtrate is seen to be opalescent and is tested as follows :

1. On boiling no precipitate is formed, but the fluid becomes turbid.
2. There is no change with acetic acid.
3. The fluid becomes thick and of a yellowish tint when treated with acetic acid and ferrocyanide of potassium.
4. There is change to a violet color when treated with concentrated sulphuric and acetic acids.

Some observers differ from the above statement in their description of the fluid of ovarian cysts; all agree as to the large number of cell elements. At one time it was thought that the fluid contained a special cell, but this is now disregarded. In rare cases the specific gravity may be lower than that of the fluid of ordinary ascites. A fluid of low specific gravity with a small amount of albumin is said to be characteristic of a cyst of the broad ligament.

Cystic Kidney. The fluid of the cystic kidney can be recognized by the properties it derives from the renal secretion. Urea and uric acid in large amounts point to its true source. Renal epithelium is of the greatest diagnostic value (see Urine). If epithelium from the urinary tubules can be detected after the fluid has settled the diagnosis is absolute (see Hydronephrosis). It must not be forgotten that both urea and uric acid may be found in other cysts, as of the ovary, if they communicate with the urinary tract.

Pancreatic Cysts. Recently the fluid from cysts of the pancreas has been examined and proved of diagnostic value in determining the nature of the abdominal tumor. The fluid is of a specific gravity of 1012, but may be as high as 1028. It contains cholesterin crystals in abundance, and blood or pigment. Serum albumin is present, but metalbumin is not found. The diastatic ferment is present. This may be met with in the fæces and in the secretions of the mouth. If on examination for sugar the latter is found to be a maltose its presence is of diagnostic significance.

The most pronounced property of the pancreatic fluid, and that by which we are enabled to distinguish it from other fluids, is the power of digesting albumin without the presence of an acid.

Boas (*Deutsche med. Wochenschr.*, 1890, Bd. xvi., p. 1095) developed the method of examination. The fluid is to be added to milk ; after the casein is precipated the biuret test is applied. Heat the substance with caustic potash and add drop by drop a 10 per cent. solution of sulphate of copper. If digested albumin is present the fluid assumes a reddish-violet color. No other cystic fluid can dissolve albumin in the alkaline solution. The fluid also emulsifies fats. In large cysts, however, particularly of long standing, the physiological properties of the pancreatic juice are sometimes wanting. In the case referred to by Boas and reported by Karewski, the old age of the cyst modified the character of the fluid and hence rendered its nature doubtful. Moreover, in the exploratory puncture the stomach was penetrated. For two reasons the author advises against exploratory puncture. First, the age of the cyst is not known, hence an analysis would be misleading. Second, the danger of puncturing other organs is too great. Exploratory laparotomy is preferable.

CHAPTER VI.

THE MORBID PROCESSES AND THEIR SYMPTOMATOLOGY.

Knowledge of symptoms of morbid processes essential; they control conclusions drawn from data.—Morbid processes are few. I. Alterations in blood and circulation: Anæmia and plethora—Hyperæmia, active and passive—Œdema and dropsy—Thrombosis and embolism—Hemorrhage—Blood-pressure. II. Disturbances of nutrition: Inflammation—Gangrene and necrosis—Fever—Atrophy and hypertrophy. Degenerations: Albuminous—Fatty—Colloid—Mucous—Pigmentary—Calcareous—Amyloid—Fibroid. III. Anomalies of growth: Tumors—Cysts—Cancer.

NOTWITHSTANDING our having secured the data obtained by inquiry and data obtained by observation based upon which the diagnosis if possible is made, the conclusion arrived at is often not final, and perhaps from the nature of the case cannot be. We are prompted, therefore, to view the case from a different standpoint, to utilize our knowledge as to the phenomena of morbid processes, and for the purpose of comparison to review the features of those that are apparently of the nature of the process under consideration. Thus, for instance, in an obscure case of fever, the objective and subjective phenomena have been fully inquired into: we are unable to decide whether the disease under consideration is a septic process with obscure lesions, a form of miliary tuberculosis, or of malignant endocarditis. The symptoms of each are considered, our knowledge of such symptoms depending upon our knowledge of the phenomena of the respective morbid process. Moreover, after a diagnosis is made, a review of the symptomatology of morbid processes answers as a control experiment to the conclusions that have been attained. We should also, after a diagnosis is made, compare the symptoms of the process in the individual case with the symptoms which we know to be of common occurrence in the disease thought to be present.

It is necessary, therefore, that the student should fully know the symptoms of morbid processes. Each process is characterized by phenomena common to it, and by which it is recognized. The symptoms are modified by the function and anatomical structure of the organ in which the process takes place. Thus the symptoms of inflammation of the mucous membranes of the bronchial tubes and of the stomach are the same except that from difference in function in the one we have cough; in the other, vomiting. Very frequently the symptoms differ because of physical and hence mechanical alterations. Thus an inflammation of the pericardium and of the pleura are allied, but in the former pressure symptoms ensue that are infinitely different, because of the anatomical relations, from the pressure symptoms of the latter.

The morbid processes are not many. They include: I. Alterations in the blood and circulation; II. Disturbances of nutrition; III. Anomalies of growth.

I. Alterations in the Blood and Circulation. The composition and distribution of the blood affects all the tissues for weal or woe. The quantity of the blood alone will be referred to; changes in quality will be considered under diseases of the blood. Practically the symptoms produced when the quality is affected are those of *anœmia* plus the symptoms (physical and functional) of the primarily diseased organ —as the spleen in leucocythæmia. The quantity may be increased or diminished.

1. Increased Quantity of Blood, or Plethora. Formerly this was considered an entity, and the symptoms of flushed face, hot and full head, throbbing pain, throbbing temporals, a full, strong pulse, sluggish intellect were thought to indicate an excess of the general bulk of the blood. True plethora is rarely permanent. If transitory, the veins and not the arteries are overfilled. The symptoms are not due to general plethora but to excess of blood-pressure or to special determinations of blood to superficial vessels, determined by a nervous mechanism. Increase in one of the cellular elements of the blood, the leucocytes, is not a plethoric condition.

2. Diminished Quantity of Blood, or Anæmia. Anæmia embraces diminution of the bulk of the blood, or any one of its morphological constituents.

The term might be used for loss of water of the blood, as in cholera Asiatica (see Infectious Diseases), or in serous purging. The symptoms are those included in the term *collapse*.

Oligæmia or spanæmia are terms that may be used to define the general thinness or poorness—atrophy of the blood. Clinically, anæmia is divided into simple anæmia, general poverty of blood; pernicious or idiopathic anæmia, reduction in the number of red cells; chlorosis, reduction in the quantity of hæmoglobin; leucocythæmia, relative loss of red, increase of white corpuscles. (See Diseases of the Blood.)

3. Local Disturbance of the Circulation. Hyperæmia, or Congestion. The process may be acute or chronic. It is usually local, although it may be general. When the latter, many organs may be simultaneously involved, due to a common cause.

Symptoms. The acute or active form of hyperæmia is always local and arterial. There is increased blood in the part. If the skin is the seat, there is redness and increased heat, and throbbing or pulsation may be seen. The parts are swollen. The excitability of the nerves is increased, with local symptoms of warmth, fulness, or itching. The morbid blushing, or flushing, that occurs at the menopause or reflexly from internal disorder, is a hyperæmia, while in erythema of the skin it is seen in most marked degree.

Causes. Arterial hyperæmia is caused by (1) neuro-paralysis of the inhibitory or vaso-constrictor fibres, of the cervical, sympathetic, splanchnic, and other sympathetic and some mixed nerves, as the sciatic; (2), neuro-tonic stimulation of the actively dilating or vaso-dilator nerves,

as the chorda tympani. Under both circumstances there is relaxation of the arterial walls. This may also occur directly through the vasomotor system, induced by heat, electricity, or chemical irritants, or from paralysis of muscular fibres, after spasmodic contraction due to cold, as frost-bite.

Causes and Symptoms of Neuro-paralytic Hyperæmia. A tumor pressing upon the cervical sympathetic nerve, abscess which destroys it, and wounds of it, produce hyperæmia of the side of the face, rise of temperature, and contraction of the pupil. Later on, the vascular conditions are reversed. Lesion of the fifth nerve, or a branch, causes hyperæmia of the iris, the conjunctiva, the cheek, the gums, and other structures supplied by it, with associate loss of sensation followed by atrophy. The latter conditions have nothing to do with the vascular paralysis.

Neuro-tonic Hyperæmia. After wounds of the brachial plexus, hyperæmia of the fingers is seen. (See Fingers.) The local temperature is elevated, and there is neuralgic pain. Local hyperæmia with hyperæsthesia, known as erythromelalgia, belongs to the same class, due to affections of the nerve trunks, or the peripheral nerve-endings. Reflex hyperæmia must be remembered.

Chronic or Venous Hyperæmia (passive congestion). The blood accumulates in the veins and, by backward pressure, in the capillaries. The venous capillaries are overdistended and, compared with the arterial, much enlarged. They contain venous blood.

Any congested part, as the exterior, is bluish or purple in tint, often swollen (clubbed fingers), cooler than normal, with lessened sensation and without pulsation. (See Cyanosis.) The dependent parts are first affected, as the legs, or the lungs. In fevers the weak heart and the recumbent posture predispose to the latter.

Causes. Obstructive heart and lung disease cause *general* venous congestion. Local venous congestion is caused by tumors, the pregnant uterus, collections of fæces pressing upon the veins. It is also caused by inflammation of the veins, as thrombosis.

Local Anæmia. This may be due to arterial thrombosis or embolism; arterial obstruction through endarteritis; arterial spasm. Raynaud's disease is a form of arterial spasm. The grave effects of arterial obstruction are seen in cerebral anæmia from endarteritis, or myocarditis from obstruction of the coronary arteries.

Œdema and Dropsy. The changes of the circulation which produce these conditions have been referred to in the third chapter of this book. The symptoms and signs of the condition are also noted in the same section.

Thrombosis and Embolism. The student should be familiar with the symptoms of these conditions and, fully as important, with the causes that give rise to them. Thrombi may form in the heart, the arteries, or the veins. Emboli may form in either vascular channel, but are found in the vessels only.

Thrombosis. The symptoms of thrombosis are: 1. *Mechanical.* The channel is obstructed; hyperæmia, engorgement, œdema, and cyanosis arise. Its most typical form is seen in femoral thrombosis, with swelling

cyanosis, and œdema of the leg. When an artery is obstructed, the symptoms are like those of occlusion under other circumstances (see Embolism); when a vein, the mechanical symptoms vary in accordance with the particular vein affected. Thus in thrombosis of the coronary vein, the heart's action is interfered with; of the portal vein, jaundice (not because of the obstruction), œdema (ascites), congestion (gastric and intestinal) occur, as in obstruction in any vein; in the cerebral veins, disturbance of the function of the brain; in the pulmonary veins, dyspnœa. 2. *Inflammatory or septic.* If it should happen that the thrombosis developed secondarily to an inflammation of septic origin, as in the extension of an inflammation into the radicles of the portal vein from an abscess about the rectum or vermiform appendix, it would be infected with micro-organisms. An infectious inflammation with chills, fever, sweats, and other phenomena of a septic character would result. 3. *Embolic.* From the thrombus, emboli are washed off; hence, embolic symptoms arise in the course of thrombosis.

While thrombosis is usually easily recognized, it is necessary to call attention to the very great importance of going a step beyond, to seek for the cause of the thrombosis. Knowing the causes of thrombosis, often a thrombus otherwise not suspected can be adjudged as the cause of the symptoms. The causes are not many. 1. Stagnation or stoppage of blood. It is seen chiefly in the veins and the heart. External pressure upon the veins, as upon the pelvic veins in pregnancy or abdominal tumor, upon the hemorrhoidal veins, upon the portal veins by tumor, upon the pulmonary veins by mediastinal tumor. It must be remembered that some change takes place in the internal coat of the vein also, but that the pressure is primary. Then we have weakness of the heart as a cause of stagnation. Feeble contractions lead to the formation of cardiac thrombi. 2. Thrombosis from changes in the walls. The change is usually inflammatory and often proceeds from wounds. If the wound was septic, the inflammation will be septic. In the heart, endocarditis; in the aorta, atheroma leads to the development of thrombi. 3. Thrombosis from the entrance of a foreign substance into the vessels. A carcinoma or other new growth may extend into the veins. Micro-organisms penetrate the vein and cause inflammation and thrombosis, or infect a previously existing thrombus. The clot is then broken and distributed throughout the system, causing pyæmia. 4. Thrombi are produced by extension. A clot enlarges by coagulating the blood next to it. A large vénous distribution may become blocked, as first the uterine veins, then the internal iliac, then the external iliac, and from thence the femoral—causing the affection which frequently occurs in the puerperal form, phlegmasia alba dolens.

Embolism. An embolus is a substance which plugs a vessel. It may be a fragment of blood-clot (thrombus), vegetations from valves of the heart, parasites, new growths which entered the veins, fat, or air. If obstruction of the vessel alone is produced the embolus is said to be simple. If obstruction and a new process, as inflammation, is set up it is specific. Fragments from a thrombus in the systemic veins may produce an embolus which blocks the pulmonary artery; a clot or portion of valve leaflet from the left heart will block a systemic artery, as

a cerebral vessel or the femoral vessel; a clot in the portal vein will obstruct branches in the liver.

The symptoms are sudden in occurrence and depend upon the artery that is obstructed. The cutting off of blood supply causes, beyond the point of obstruction, cessation of function. In pulmonary *venous* embolism the dyspnœa is pronounced, the heart's action rapid and irregular, and many cases are called cases of "heart failure." In the middle cerebral artery the embolus causes aphasia and mono- or hemiplegia. In embolus of the pulmonary artery cough and hemorrhage with dyspnœa occur suddenly. The patient has, in this instance, no doubt, mitral regurgitation and dilated right heart.

The result of blocking of an artery varies in many cases. If the main artery of the leg is blocked, anastomosis may be set up to compensate for the obstructed channel. If this is not secured gangrene ensues. If an artery to an internal organ is blocked anastomosis may occur, if the artery is not terminal. If the artery is terminal there results rapid necrosis or softening, as in the brain; gradual wasting, as of a kidney, or engorgement of the arterial area and diffuse hemorrhage. The latter is known as a *hemorrhagic infarct.* This may occur in the lungs (pulmonary artery), spleen, kidneys, retina, and, rarely, the intestinal canal. The symptoms of hemorrhagic infarct are swelling and hemorrhage. In the lungs, there are physical signs of consolidation, with hæmoptysis, cough, and dyspnœa; in the kidneys, pain and hæmaturia; in the spleen, pain and at times enlargement; in the retina, blindness with ophthalmoscopic changes; in the intestine, pain and hemorrhage with sloughing of mucous membrane. *Infective emboli* cause abscesses. *Capillary embolism* is seen in the skin and mucous membranes in many infective diseases, notably ulcerative endocarditis. *Fat embolism* occurs in the pulmonary capillaries, and is due to fat globules which sometimes enter the circulation in pregnant women, in patients with bone disease, as osteomyelitis or fractures. The symptoms are those of intense dyspnœa. It may cause sudden death.

Air Embolism. Air may enter wounds of the veins of the neck. It accumulates in the heart, and as the ventricle cannot contract on it the blood is not propelled. Death takes place with the symptoms of heart clot, the heart being in asystole.

Hemorrhage. Hemorrhage may be arterial, venous, or capillary. It may occur because the blood soaks through the walls, by diapedesis; or it may occur from rupture, or rhexis. The former takes place in venous engorgement, stasis, or inflammation. It is the small passive hemorrhage of congestion, as in pulmonary congestion from heart disease; it is venous or capillary; the blood is dark. Hemorrhage by rupture is arterial, venous, or capillary. If the artery, it has been torn by violence, destroyed by ulceration or suppuration, or it is the seat of endarterial change. Veins are also diseased or the walls destroyed before rupture takes place. Rupture of capillaries occurs from violence or great internal pressure. In death from suffocation the capillaries are the seat of hemorrhage because of the increased venous pressure. It occurs in typhus, hemorrhagic smallpox, and scarlatina. The state of the blood sometimes is the cause of hemorrhage, as in scurvy,

purpura, and other conditions. Hæmophilia is a peculiar hereditary affection due to the state of the blood possibly—more likely to the condition of the bloodvessels.

The special forms of hemorrhage and their symptoms, ætiology, and diagnosis will be considered in the sections to which the name in the following list points :

Bleeding from the nose—*epistaxis.*

Vomiting of blood—*hæmatemesis.*

Bleeding from the lungs—*hæmoptysis.*

Blood passed with the urine—*hæmaturia.*

Blood passed from the uterus—*menorrhagia* or *metrorrhagia.*

There is also intestinal hemorrhage—*melæna.*

Hemorrhages underneath the skin are known as *petechiæ* if small, and *ecchymoses* or *suffusions* if large.

Hemorrhage into internal organs receives its name from the organ affected and is known as a *parenchymatous hemorrhage. Apoplexy* is applied to hemorrhage into the substances of organs, particularly if it occurs suddenly and is localized—as pulmonary apoplexy, cerebral apoplexy, spinal apoplexy. Long usage has associated the term with hemorrhage into the brain, so that it is applied to that form only by most writers. *Hæmatoma*, or blood tumor, is a collection of blood that has coagulated in a cavity, organ, or tissue. (See Ear.)

The *symptoms* of hemorrhage vary in degree in accordance with the amount of blood which escapes from the vessel, and depend upon whether the hemorrhage is external or internal. By an external hemorrhage we mean one which is accompanied by a discharge of blood visible to the bystander. An internal or concealed hemorrhage is not apparent by any outward sign of blood.

The symptoms by which *external* hemorrhage is recognized need not be detailed. The show of blood in situations or at times other than normal is sufficient. It must be remembered that arterial blood is bright red, venous blood dark. It must also be remembered that the character of the blood from internal organs is modified by the secretion of the affected organ. Thus the blood from the stomach is black, coagulated, like coffee-grounds ; from the intestine, tarry. The general symptoms of the various degrees of external hemorrhage are similar to the symptoms of internal hemorrhage, which will be described later. Both vary with the rapidity of the flow of blood. If the bleeding is slow large quantities may be lost and give rise only to more or less profound anæmia. It is more difficult often to determine the source of hemorrhage. The mode of recognition of the anatomical varieties of hemorrhage will be discussed under the respective systems which are the seat of the bleeding. Hemorrhage may take place in a cavity, as the stomach, bowels, or bladder, and after it has undergone changes cause symptoms of, and be discharged as, a foreign body.

Internal hemorrhage presents vivid phenomena. The recognition of the hemorrhage is often impossible without some knowledge of the history of the case, as will be spoken of later. The symptoms are complex. First, there is the symptom due to rupture of the vessel or to the filling of a tissue with blood—that is, pain. In the first instance it is

sharp, severe, and of itself may cause shock. In the next place, the symptoms due to loss of blood arise. After pain, sudden prostration ensues; pallor spreads rapidly; the extremities become pallid; they cool and later become cold; a cold sweat breaks out on the forehead; the features become pinched and shrunken; the pulse becomes weak and rapid, and later thready, or disappears at the wrist; the carotid pulsates; the heart throbs violently and a diffused impulse is seen, at first vigorous, soon like a slap against the chest wall, and then it fades away completely. On examination of the heart and vessels, so-called anæmic murmurs are heard. The patient is restless and tosses to and fro. He sighs and yawns frequently. The respiration becomes slow and shallow. Nausea and at times vomiting may occur. He may faint at once, or repeatedly, to be restored again and again, or soon fall into syncope, restoration from which does not take place. In the intervals between the syncopal attacks the mind is clear. When profound shock occurs there is dulness or stupor; the intellect is dazed. Delirium and agitation may, on the other hand, be present. When the hemorrhage is profuse, convulsions may take place. The temperature of the body falls. If the patient has fever at the time, the temperature suddenly falls to or below the normal. It is thus seen that, in hemorrhage, the conditions syncope, shock, collapse take place. They may all occur in the same subject, or one or two may be absent. They may occur from other causes, which must be excluded. Sometimes the shock produced may be due to the cause which also produces the hemorrhage. The causes of shock are so potent that they serve to distinguish it from the collapse of hemorrhage. They are injury, anæsthesia, railway accidents, surgical operations, perforative peritonitis, strangulated hernia, intestinal obstruction, profound mental impression, or pain.

Shock from hemorrhage must be distinguished from concussion. In the latter the intellectual disturbance occurs at once and is more predominant than circulatory symptoms. The absence of the usual phenomena of hemorrhage serves to distinguish syncope due to that cause from that due to the many well-known causes of fainting.

The forms of internal hemorrhage sufficient in amount to have a probably fatal result, or at least to create alarming symptoms, are many. In the chest, diseases of the lungs or the aorta cause hemorrhage. In concealed pulmonary hemorrhage the blood accumulates in a large phthisical cavity. When the aorta or an aneurism ruptures, the blood may enter the mediastinum or the pleura. Under these circumstances the previous history is essential. Careful examination of the lungs, in a case which presents the above-mentioned symptoms of internal hemorrhage, or of the heart or bloodvessels, must be made. Internal concealed hemorrhage into organs or cavities of the abdomen occurs in gastric, duodenal, or intestinal ulceration; in aneurism or in ulceration of large vessels, from septic inflammation about them. It must not be forgotten that alarming or fatal internal concealed hemorrhage may be due to hæmophilia or purpura.

II. **Disturbances of Nutrition.**

HYPERTROPHY AND ATROPHY. (See Size, and Muscles.)

INFLAMMATION. Inflammation is a process largely attended by vas-

cular alteration, but also with disturbance of nutrition. It may be acute or chronic. It is due to injury, mechanical, physical, chemical, or vital. The invasion of micro-organisms or the irritation of their products is the most frequent cause in cases that come within the province of the physician. The symptoms are modified by the structure affected and the cause of the inflammation. The intensity and the character also modify them. The classical symptoms—*pain*, *heat*, *redness*, and *swelling*—are symptomatic of the tissue process. In addition, we must add *exudation* and *alteration of function*. *Pain* varies in degree with the sensibility of the part. When accessible, it is increased by pressure or movement, or by the functional activity of the affected organ. *Heat* is detected by the hand or surface thermometer. It may be complained of by the patient, in abscess within the peritoneum or pyosalpinx, as a ball of fire. The temperature over an inflamed lung or pleura is higher than over the healthy side. *Redness* can only be observed in parts open to inspection, as the nasal, oral, faucial, and other cavities. *Swelling* is observed with the redness or detected by enlargement of the affected organ, if it can be measured by palpation or percussion. *Exudation* takes place from mucous surfaces, into serous cavities, into the connective or any affected tissue, into tubes or channels (heart and bloodvessels, lymphatics, etc.). The exudation gives rise to symptoms. Characteristic discharges from mucous surfaces; pressure and physical signs from accumulation into cavities; symptoms due to the obstruction of channels; grave pressure symptoms when impinging on nerves, on nerve centres, or nerve tracts (brain, cord, peripheral nerves) are due to exudation. The pressure symptoms are often more pronounced than the inflammatory in simple or tuberculous meningitis. *Alteration of function:* The symptoms cannot be detailed; each organ and structure must be referred to. The function may be stimulated at first, but is soon perverted, or suppressed.

General Symptoms. Fever is the general expression of the local process. It may be primary from reflex irritation of afferent nerves which influence the heat centre and disturb the thermo-taxic mechanism. It may be secondary; the products of inflammation (pus, toxins, etc.) irritate the centres. The degree depends upon the cause. Considerable inflammation may occur without fever.[1]

Suppuration. The character of the fever indicates the variety of the inflammatory process. In most inflammations the fever is continuous. When suppuration commences or is present it becomes intermittent or remittent. The presence of suppuration is also made known by *hectic*. The fever is attended by chills and sweats. In suppuration there is leucocytosis. The appetite is lost or impaired. The urine contains a large amount of indican. The latter tests may be of service in determining the nature of an inflammation about the peritoneum. Apart from the temperature range, the symptoms of fever are not modified by the process, save in degree. Septic inflammations are attended early by cerebral symptoms, prostration, and the typhoid state.

As a corollary, when the symptom fever is present, local inflammations must be sought for. *Chronic inflammations* may only give rise to altered function and cause exudation (swelling, effusion, etc.).

[1] Musser: "Abscess of Liver," Univ. Med. Magazine, 1892.

Inflammation of Various Structures. The symptoms are modified by the structure.

Inflammation of *mucous membranes.* *Pain* is not excessive; *heat* is complained of (rectum); *redness* is marked and varies with the intensity from bright to dark red; *swelling* is always present. In narrow channels, as the nose, or the gall-ducts, it causes occlusion. The *exudation* is at first mucous, then muco-purulent, and then purulent. Before exudation there is a stage of dryness. The microscopical appearance of the exudate varies with the anatomical character of the membrane affected. Its peculiar epithelium is always present, micrococci, pus, red cells; from the lungs or liver, special crystals. The functions are impaired. Fever is usually not very high; it is continuous. The causes are diret local irritants or congestions from external impressions (cold?).

Inflammations of *serous membranes.* *Pain* is extreme and may cause collapse. *Heat, swelling,* and *redness* cannot be estimated. The surface temperature is raised. *Exudation* occurs after a brief dry stage. The cavities—pleura, pericardium, peritoneum, joints, cerebro-spinal canal—are filled, causing mechanical symptoms and physical signs. Fever is excessive in some forms, depending on the cause. Function is impaired or abolished. General symptoms are more pronounced. Shock or collapse is common in peritonitis. The affections are always secondary to a general process (rheumatism), to infection, to disease of neighboring structures, or to Bright's disease, diabetes, cancer, scurvy, or other diathetic condition.

Inflammation of *muscles* (rare), of *connective tissue,* and of *glands* are characterized by symptoms common to the morbid process, with alteration of function.

Inflammation of *bone* and *periosteum* presents the same group of symptoms. The pain may be intense or of a dull aching or boring character.

Inflammation of the *heart* and *vessels* is also attended by the cardinal symptoms. Pain, when the central organ is the seat of the disease, is not common, but in the arteries or veins is of frequent occurrence. The striking symptom, however, is the *obstruction* to the channels. It is characteristically seen in phlebitis, as of the femoral vein. Œdema of the leg, and cyanosis, tell of the obstruction. In the heart, the acute process or the results of the process lead to the occurrence of all the symptoms of obstructive heart disease.

Inflammations of the *nerves,* the *spinal cord,* or the *brain* are followed more strikingly by pressure symptoms and the symptoms of the secondary degenerations of the inflammatory process. Hence, while pain and tenderness are present in the exposed nerves, abeyance, perversion, or abolition of function are the principal signs of inflammation of these regions.

Inflammation of *internal organs, lung, liver, kidneys,* and *pancreas,* is made known by pain (minimum sign) and swelling (enlargement of liver), and by change in the function, indicated by modifications of the respective secretions as well as by functional or physiological symptoms.

Local Death, Necrosis, and Gangrene. If nutrition is not complete, the life of the cells is endangered; they soon die. The processes

are known as necrosis or gangrene. The nutrition is annulled: 1. By stoppage of the circulation; 2. By the direct action of an irritant which destroys the cells; 3. By abnormal temperature. A combination of the three causes produces gangrene quickly. Stoppage of the circulation may be due to an embolus or thrombus, or to stagnation by pressure, or, independently, in capillary stasis. Sloughing and "bedsores" ensue in the latter instances; gangrenous eschars in the former. The cells are destroyed by corrosives and caustics, by heat and cold, by bacteria. Where decomposition takes place, as in retained and infiltrating urine, cell destruction and sloughing ensue. All pathogenic bacteria cause necrosis to greater or less degree. Frost-bite and burn illustrate the destructive power of abnormal temperature. The symptoms are local.

Nerve lesions, trophic disorders, produce necrosis. Decubitus is a form of necrosis which arises in spinal cord diseases. The sloughing is extensive and rapid. Trophic disorders cause paralytic hyperæmia, and hence necrosis.

The part that debility, cachexia, and feeble circulation play in assisting the local changes must not be forgotten.

Gangrene of internal structures concerns us. Such form is nearly always due to stoppage of the circulation. It is seen in constriction of the intestine, from hernia, or obstruction. It occurs in phthisis from thrombi. Clinically, we see it frequently in diabetes. The lung, the brain, the intestines, are most frequently affected.

The symptoms of necrosis or gangrene are modified by the tissue and function involved. If external, the decomposing structures emit a foul odor, there is rapid prostration and the development of the typhoid state. Fever ensues because of the intoxication by decomposing substances—sapræmia. Often the symptoms are latent. A man aged sixty in my ward was about all the time. He died suddenly of pulmonary hemorrhage, the result of gangrenous ulceration of a large vessel. At the autopsy gangrene of the lung was found. The only symptom was the characteristic odor. In the course of inflammatory processes its onset is frequently attended by the cessation of pain, the occurrence of the peculiar odor, if accessible, and the development of exhaustion and the typhoid state. The character of the discharge points to gangrene. When the lungs are affected, the expectoration is like prune juice; when the bowels, the discharge is dark and putrid.

Fever is a morbid process, with the cause and symptomatology of which the student must be familiar. It has been fully treated of in a preceding section. (See Fever and Infectious Diseases.)

The Degenerations. The symptomatology varies with the form of degeneration and the organs affected. The general economy is in a state of prostration for the same reason that the degenerations are present. *Albuminous* degeneration occurs in fever, and causes the weak heart and defective gland action. The weak heart of the convalescent period in diphtheria and other infective diseases is well known.

Fatty Degeneration. *Fatty infiltration or lipomatosis* is seen in the "fat" heart of brewers, the enlarged liver, the excess of fat in the abdomen, etc. The affected organs are enlarged, they are functionally weak. Fatty infiltration of organs is recognized by its ætiological asso-

ciations. If with the above conditions the liver is enlarged or the heart weak, or both, we may expect to find this degeneration. There is enlargement of the affected organ, which is painless, smooth, not usually soft on palpation. The condition occurs at any age, but usually in later life. Emaciation may not occur. Lithæmia is common in fatty infiltration. In alcoholic subjects living sedentary habits, in subjects who eat an excess of fatty foods, in overfed and pampered children, and in tuberculosis, it is commonly seen. In fatty infiltration the cells are not destroyed. In fatty degeneration there is cell destruction. The brain, the heart, the kidneys in Bright's disease, the liver, all undergo degeneration. It may be due to phosphorus poisoning or snake-bite. It is seen in acute yellow atrophy of the liver.

Amyloid Degeneration. This is rarely confined to one organ of the body; many are affected. The causes are syphilis, malaria, tuberculosis, and prolonged suppuration. The *liver* and *spleen* are *enlarged*, hard, smooth and painless. There is great *pallor*, œdema of the feet and face. There is anæmia, but no fever. The kidneys are affected, hence polyuria and low specific gravity of the urine; a few casts are found. The bowels are likely to be loose from degeneration of the intestinal walls. It occurs at any age. The diagnosis rests on the presence of a cause, the painless enlargement of organs, the pallor, the polyuria.

Fibroid Degeneration. This is not a degeneration, but rather an overgrowth of connective tissue with coincident primary or secondary atrophy of the parenchyma. The function of the organ is impaired or abolished. The increase of connective tissue in the nerve structures is known as sclerosis, in the liver or kidney as cirrhosis. In the artery it leads to the changes known as endarteritis. Whatever the pathology may be, particularly as to the question whether atrophy of cell elements of the affected structure is primary or secondary, nevertheless the condition is productive of serious, even grave, symptoms. It is part of the senile process. It leads to the manifold symptoms of endarteritis; it is the cause of many nervous affections which will be treated of in their proper sections. The varied phases of so-called interstitial nephritis are due to the fibroid change primarily in the kidneys, and secondarily in the arterial system. In the lungs it attends emphysema, and may be productive of that condition. The fibroid heart arises because of it. Tubes and channels are closed by the same process as in fibrous stricture of the duodenum. Wherever situated its development means gradual abolition of function.

Mucous Degeneration. This form of degeneration is seen in myxœdema, previously described. The albuminous intercellular substance is replaced in the connective tissue by mucin.

Pigmentary, calcareous and colloid degenerations are local morbid processes without symptoms beyond those due to the primary affection.

Tumors and New Growths. Tumors other than cancer or sarcoma, produce symptoms only mechanical and must be considered in their special section. They are due: 1. To the tumor (foreign body). 2. To obstruction of any channel in near relation. Cancer and sarcoma are accountable for a group of symptoms to which the term *cachexia* has been applied.

It is true they produce local symptoms. This is most striking when the growth develops in structures which must be destroyed from increase in its size, as in the brain or spinal cord, or where tubes or channels may be closed, as in cancer of the stomach or œsophagus. (See Special Diagnosis.)

Local symptoms may precede the general symptoms. On the other hand general symptoms may arise, the local cause for which cannot be discovered. The local symptoms of cancer of any particular organ are variable and dependent upon the anatomical nature and physiological offices of this organ and upon their anatomical relation with surrounding organs. This class of symptoms will be referred to in the section on special diagnosis. They are symptoms due to gradual abolition of the function of the organ, and closure of the channels in connection with it, as the intestinal canal, the pharynx, or the hepatic ducts. A few symptoms are common to the cancerous process. They may or may not be present; in the large majority of cases one or more are present; they should always be sought for in order to confirm a diagnosis of cancer.

1. *Pain* is a common symptom, recognized by peculiar characteristics in most cases: (*a*) It is sharp and lancinating; (*b*) it is paroxysmal; (*c*) it is increased by irritation, as food when the stomach is affected; (*d*) it is increased by functional activity, especially if movement is excited, as speaking or swallowing in carcinoma of the larynx or pharynx respectively.

2. *Hemorrhage* is a common occurrence in carcinoma. If the malignant mass is in communication with the exterior by channels, the blood may be discharged *per vias naturales*. In malignant disease of the upper air-passages or in the lungs, hemorrhage is likely to occur. In gastric carcinoma it is common. Its occurrence in uterine cancer is well known. If the organs are not connected with the exterior, but give rise to exudations or transudations, the latter are frequently bloody, as in carcinoma of the pleura or peritoneum.

3. *Abnormal Discharge.* This particularly occurs in cancer of the hollow viscera; of the canal structures. The discharge is the result of inflammation, suppuration, and necrosis, and particularly microbic inflammation. It is recognized by its more or less *bloody character* and by its *odor*. The latter is peculiar. It is most offensive and penetrating, and, particularly in uterine cancer, is almost pathognomonic. Even with the utmost attention to cleansing, it cannot be removed.

4. *Tumor.* It may be readily detected or elude all search. Some swelling is certainly present. It is recognized by external examination, by the objective physical signs of enlargement or change of contour of the affected organ.

5. *Foreign body.* The growth gives rise to symptoms, similar to those that are present when a foreign body is fixed in any portion of the hollow viscera, as the respiratory tract, the gastro-intestinal, including the hepatic and the genito-urinary tract. *a.* Through reflex influence an attempt is made by the patient for its removal, hence cough, vomiting, diarrhœa with tenesmus, repeated and painful micturition with tenesmus, etc., the particular symptoms varying with the organ

affected—are induced. *b.* Obstruction of the channels, with the many symptoms that arise therefrom, depending upon the location of the growth.

6. *Temperature.* A morbid process is often recognized by its negative symptoms, if the term may be used. Thus, fever is absent or the temperature is subnormal.

7. *The Cancerous Cachexia.* Wherever situated the disease sooner or later is attended by extreme general symptoms which are in a measure striking. It is to be admitted that cases of carcinoma often occur without marked cachexia. *a.* One symptom may always be looked for; it is *emaciation.* It may be rapid or may be gradual and extend over one to two years; toward the end it is always rapid. Ultimately, if the patient does not succumb to other conditions, it presents an extreme picture. The eyes are sunken, all normal accumulations of fat disappear. The fat in the rectal fossæ disappears, causing deep depression of the rectum. The abdomen is retracted. The appearances are probably more striking in cancer of the œsophagus than in any other organ. *b. Pallor*—a peculiar hue is seen (see Facies); this may be absent. *c. Anœmia,* with breathlessness, palpitation, vertigo. *d. Exhaustion.* This progresses with emaciation. It may be the first symptom. Progressive weakness, without fever or local disorder to account for it, is often seen. Toward the end it becomes so extreme as to forbid exertion. *e. Malnutrition.* Evidences of malnutrition are seen. The skin is hard and dry. Its elasticity is impaired. It becomes the field for parasitic invasion. Tinea and other parasites may flourish. Bacteria invade the susceptible areas; boils occur in full degree. The secretions are perverted. In the mouth ulcers develop; the fungi of this situation (throat, etc.) become more active; the gums are inflamed. In the later stages the "typhoid state" (see Fever) may ensue. If the gastro-intestinal tract is invaded symptoms of acute intoxication may arise.

8. *Metastasis.* We are often aided by the occurrence of this event, particularly glandular involvement. In gastric carcinoma, evidences of secondary hepatic disease or enlarged glands above the left clavicle; in rectal carcinoma, secondary hepatic cancer points to the primary process most conclusively. In many instances the presence of cancer is recognized by the metastasis even when the primary growth cannot be recognized.

The *diagnosis* rests upon the above conditions. In obscure cases, the age, the sex, the associate pathological conditions, the duration of the disease, are of service. Cancer usually occurs after forty, or some authorities say, after fifty years of age. The female sex is most frequently affected. It may be associated with a history of previous lesion or irritation, as ulcer in vaginal, gastric, or rectal cancer; the irritation of teeth or a pipe in labial and lingual cancer; of gall-stone, in cancer of the bile-ducts; of renal or visceral calculus in disease in that situation. A disease of grave and malignant character, the duration of which is over eighteen months or two years, is not, in all probability, cancer.

Morbid Processes in Tubes or Channels. *The effects produced by obstructions.*

The morbid processes previously described have nothing to do with the lesions of the anatomical structure which makes up the channel. Reference is to be made only to obstruction of the canals. The symptoms derived from obstruction of the bloodvessels and lymph channels, cyanosis, œdema, gangrene (thrombosis and embolism), have been described. Sufficient stress was not laid upon one secondary symptom group, a group which arises in obstruction of all channels. The symptoms are due to hypertrophy in front of the point of obstruction. In the cases of vascular obstruction the hypertrophy is seen in the heart and the arteries. (See Diseases of the Heart.)

In obstruction of other tubes or channels there is observed in more or less degree, first, hypertrophy in front of the obstruction; second, regurgitation, damming up of material which normally passes through the channels; third, atrophy and cessation of functional activity beyond the point of obstruction; fourth, cessation of the flow of the normal fluid along the canal obstructed; fifth, dilatation of the hypertrophy which took place primarily; sixth, degeneration, ulceration, low-grade inflammation (bacterial), secondary rupture of the affected viscera. The morbid anatomist can readily point out examples of the morbid changes that are sequential to obstruction. Thus in cancer of the œsophagus there is hypertrophy of the muscular coats, regurgitation of food, atrophy of the stomach, dilatation with accumulation of food, secretions from the glands of the œsophageal mucous membrane, secondary ulceration, rupture into the lungs, with gangrene or pneumonia. In obstruction at the pylorus there is, first, hypertrophy; second, accumulation, regurgitation; third, intestinal atrophy; fourth, dilatation of the stomach, with its train of symptoms. In obstruction of the biliary channels, or the bladder, or ureters, the same secondary conditions arise, plus obstruction to the flow of bile or urine. Secondary symptoms arise from accumulation of the non-escaping fluids. Subjective symptoms, it may be said, are not marked, but, if present, are pain and difficulty in the performance of the usual functions. It need scarcely be said the obstruction sometimes gives rise to symptoms which are due to the abnormal obstructing material which acts as a foreign body. The symptoms are reflex and depend entirely upon the seat of the foreign body. The symptoms are most marked when the obstruction is due to disease outside of the walls or to obstruction by occlusion within the walls.

The *causes* of obstruction in whatsoever channel situated are, first, pressure from disease outside (growths, hernia); second, disease of the walls, with contraction; third, occlusion by a foreign body, as gallstone, renal calculus, worms, or other material, depending upon the channel obstructed.

In all cases of obstruction, nasal, faucial, laryngeal, bronchial, œsophageal, gastro-intestinal, biliary, renal, or pancreatic, look for the symptoms of the secondary morbid change. Each form of obstruction will be considered elsewhere. (See Special Diagnosis.)

The Bloodvessels. Blood-pressure. It must not be forgotten that the bloodvessels are in a measure distinct from other tubes, although subject to the same laws, physiological and pathological. They contain fluids and have a continuous function by which the fluids are propelled. They are subject to the laws that govern the flow of fluids under all circumstances in nature. Any derangement or disease will cause violation of the hydrostatic or hydrodynamic laws. Fluids within vessels exert pressure. Pressure produced by the weight of the fluid is known as the hydrostatic pressure; produced by the flow is known as the hydrodynamic pressure. Pressure can be gauged and its degree ascertained by proper instruments. In the case of fluid in the bloodvessels, it is called the blood-pressure. The blood-pressure is estimated by the educated finger on the pulse and by the sphygmograph. A certain definite pressure is always present in health. It is subject to slight fluctuations, but tracings with a sphygmograph follow a definite course. In the description of the pulse, modifications of blood-pressure will be given in detail; it is sufficient here to say a few words regarding hydrostatic and hydrodynamic pressure.

Hydrostatic pressure is modified by the weight of the fluid. It is of importance in the veins only, and especially those of the lower limbs, in pathology. When the pressure is increased, first, because of increased weight, or second, because of loss of power to support the pressure (the most common), varicose veins and venous congestion arise. Inflammations of the lower limbs are attended by venous accumulation and followed by ulceration. Dropsies in these portions arise more readily for these reasons. The common occurrence of gout in the feet may arise from slowness of the circulation.

Hydrodynamic pressure is variable. Its changes indicate increase or diminution of blood-pressure. The bloodvessels are resisting elastic tubes; the resistance is always equal to the pressure within, hence blood-pressure and arterial tension are equivalent terms. We speak of increased or diminished pressure, or correspondingly of high or low tension. Now the hydrodynamic or blood-pressure depends upon, 1, variations in the volume of blood; 2, variations in the capacity of the vascular system; 3, facility of the capillary circulation; 4, the force of the heart. The tension of the artery is dependent upon the same factors.

1. Variations in the volume of the blood. *a.* Volume increased. Causes, absorption of fluid after meals or drinking more fluid. Result, increased blood-pressure and increased tension. Controlled in health by action of the vasomotors relaxing the vessels, causing their enlargement, and by enlargement of the veins. *b.* Volume diminished. Cause, hemorrhage, serous purging. Result, diminished blood-pressure, lowered tension. Controlled in health by contraction of arteries through vasomotor nerves. In hemorrhage the loss of blood produces anæmia. The latter is a stimulant to the vasomotor centre in the medulla, and produces contraction of peripheral arteries and high tension.

2. Variations in the capacity of the vessels. *a.* Diminution of the capacity of the blood channels (volume of blood not lessened). Cause: cutting off of a vascular area by ligation or obstruction, by narrowing the calibre of the walls as in arterial spasm or endarteritis, by disease of

the kidneys lessening channels in the aortic circuit, or disease of the aorta causing obstruction to the outflow of blood. Result: increased pressure, high tension. Controlled by normal regulating vasomotor apparatus, or by diminution of the volume of blood. *b.* Increase of capacity of blood channels. Cause, relaxation of muscular coats of vessels. Result, diminished blood-pressure, lowered arterial tension. Controlled by contraction of vessels or increase in amount of blood. In shock, the vasomotor sympathetic system of the splanchnic arteries is so disturbed that the arteries are dilated and all the blood is sent into the abdominal vessels (fall of pressure).

Mode of action of the vasomotor apparatus. Centres in the medulla, in the spinal cord, and in the sympathetic ganglion control the vasomotor nerves, which influence hydrodynamic pressure. 1. If the centres are stimulated, tonic contraction of the vessels is produced. This may be general or local. Increased pressure or heightened tension is the result. It may be a reflex from the periphery, or due to some state of the blood. 2. If the centres are paralyzed, or inhibited, or cut off from the arteries, the latter become relaxed (dilated). The pressure is lowered, the tension is less. *Shock*, pain, certain drugs, reflexes (probably) produce inhibition.

3. Facility of capillary circulation. Obstruction to outflow of blood from capillaries into veins increases blood-pressure. Cause, the same as when arteries contract. Result, increased blood-pressure, high tension. Regulated in same manner as arteries. Relaxed capillaries produce opposite conditions.

4. The force of the heart. *a.* Heart's action (left ventricle) increased. Cause, hypertrophy, palpitation. Hence greater force of blood impact, greater resistance by arteries. The tonic resistance narrows the calibre of the vessels. Result, increased pressure, higher tension. *b.* Heart's action weakened. Hence, less force of blood, less resistance. Result, lessened pressure, low tension.

The *recognition* of variations in tension. (See Pulse.)

1. High arterial pressure or tension. By (*a*) incompressibility and tension of the arteries; (*b*) accentuation of the aortic second sound; (*c*) prolongation of the left ventricle first sound; (*d*) increased flow of urine, pale and watery; (*e*) characteristic pulse tracing by sphygmograph. If the high tension is permanent, (*f*) hypertrophy of the heart; (*g*) atheroma, more or less.

2. Low arterial pressure or tension. By (*a*) soft, compressible, often dicrotic pulse; (*b*) enfeebled sounds, aortic second and left ventricle; (*c*) scanty high-colored urine; (*d*) special pulse tracing. If permanent, stases, congestions, cyanosis, with general weakness and impaired nutrition.

PART II.

SPECIAL DIAGNOSIS.

CHAPTER I.

DISEASES OF THE NOSE AND LARYNX.

The Nose.

THE symptoms of disease of the nose are due to the function and the structure of the organ and the morbid process which affects it. *Physiologic symptoms:* The sense of smell may be impaired, and symptoms due to obstruction of the canal occur in more or less degree in nasal affections. *Obstructive symptoms*: On account of obstruction more or less marked, retention of secretions occurs. The secretions are exposed to infection from without by bacteria. Putrefaction and fermentation occur and give rise to offensive odors. More serious is the effect of the obstruction on the rest of the respiratory tract. On account of nasal or post-nasal obstruction, the air must pass through the mouth. The patient becomes a mouth-breather, and, in addition to the change in the face that takes place, the voice changes, snoring is common, and mastication is interfered with; and, notwithstanding that the mouth is used as an air-passage, there is a diminution in the amount of air passing to the lungs. As a result a vacuum is created which is compensated for by external pressure. In children the result of this is marked deformity of the chest, leading to the development of the "chicken-breast." The sides of the sternum are depressed, the transverse groove is increased, the sternum itself is projected forward. The general symptoms that accompany such interference with breathing will be referred to again.

Symptoms due to the Anatomical Structure. The nose is an open space or series of air-spaces lined with *mucous membrane.* The mucous membrane is the frequent seat of microbic inflammation, as noted in hay fever, influenza, or measles. Most of the nasal symptoms are due to affections of the mucous membrane. This membrane is subject to affections that are common to all mucous membranes, and the subjective and objective symptoms are similar to those that arise in other organs, modified by the function and the anatomical arrangement of the membrane. The richness of the membranes in bloodvessels and glands is the cause of one of the symptoms, namely, the *discharge.* Moreover, the difficulty of removing the discharge on account of the various cavities in the nose in which they are pent up leads to putrefaction and the occurrence of

odor. Because the air is constantly passing over the parts, discharges are very liable to become dry, and hence *crusts* and *scabs* form. The *vascularity* of the structures of the nose is also the cause of the development of symptoms. The bloodvessels are richly supplied with nerves, which cause them to contract or dilate on comparatively slight provocation, by reflex action. Chilliness of the body, or of local areas of the body, chilling of the extremities, and other peripheral impressions, are followed by congestion of the nasal mucous membrane, which may go on to inflammation. The vascularity predisposes to hemorrhage. The nose is richly supplied with *nerves* (in addition to the olfactory nerve) which are susceptible of various irritations or impressions—impressions made by the air laden with unusual material, as fumes of a chemical nature, emanations from animals or from plants, and certain materials not yet isolated, which are decidedly irritant to them. A local source of irritation often occurs, by polyps and adenoid growths, and foreign bodies, or enlarged bone. The nerves are connected by a mechanism which is directly connected with the centres in the medulla, particularly so the pneumogastric. The effect of peripheral nasal irritation may be felt reflexly in the area of distribution of that nerve; hence from an unpleasant odor we may have the sudden occurrence of nausea or vomiting. But of more striking and frequent pathological significance is the occurrence of asthma, or sudden dyspnœa, on account of reflex excitation of the pulmonary division of the pneumogastric nerve.

Morbid processes in the nose are symptomatic of some general affections. We will not speak again of the occurrence of asthma, or of deformity of the chest and general ill-development. Acute inflammations are significant of the exanthematous diseases, particularly measles. An acute inflammation (as pointed out by Meigs) in which there is great obstruction of the nares, with an abundant, thick discharge, is a complication or early symptom of Bright's disease, that may portend the early onset of uræmia. Chronic inflammations may be due to syphilis and chronic infection from other causes.

The Data Obtained by Inquiry.

The Subjective Symptoms. These often cause extreme distress, but do not lead to a fatal termination. The general subjective symptoms are allied to those of the inflammations of other mucous membranes. *Lassitude* occurs when there is fever. It is a frequent precursor of rhinitis, and is pronounced in croupous and diphtheritic rhinitis; extreme prostration may attend the latter. *Chilliness* following the lassitude, or *rigor*, may occur in the same class of cases. If distinct rigors occur, an abscess in one of the cavities may be suspected, if the subjective and objective symptoms point thereto; or glanders may be present.

Fever. This occurs in the inflammations; it is never marked, and is not of diagnostic significance. It is more severe in *glanders* than in any other affection of the nares. It is of low type in *diphtheria*, and of *hectic* character when there is *abscess*. High fever, associated with inflammations of the nose point to the occurrence of one of the exan-

themata as the primary cause of the rhinitis. Foreign bodies in the nose may cause fever of an inflammatory character.

Local Subjective Symptoms. Pain varying in degree occurs in all of the acute affections of the nose. Its seat and character are of some diagnostic significance. A smarting or burning pain at the root of the nose accompanies acute rhinitis and attends post-nasal catarrh. The pain is diffuse and indefinite in dry catarrh and in diphtheria. The most severe pain occurs when foreign bodies are present in the nose and in cases of glanders and primary syphilis. Foreign bodies of a vegetable nature by swelling and germinating induce pain, which increases gradually in intensity.

Pain over the Frontal Sinus. Pain over the sinus is more severe than in the nose when there is inflammation of these cavities. It is sometimes so intense and agonizing as to cause serious general effects. Pain may also be located in the cheek on account of a secondary affection of the antrum. In disease of the nose, if the pain radiates to the *ear* the Eustachian tubes are probably involved.

Disturbance of the Sense of Smell. Anosmia and Parosmia. *Loss of smell*, or *anosmia*, occurs to a moderate degree in all the inflammatory and obstructive diseases of the nose. The intensity depends upon the degree of change in the mucous membrane. It may also be due to disease of the nerves or the olfactory centre in the brain. *Parosmia* is the perception of abnormal odors, and may be a neurosis or psychical difficulty entirely, and hence purely subjective, or there may be inability to distinguish an odor when presented to the nostril. All odors may appear the same, or agreeable odors may appear to the patient very disagreeable. In addition the patient may complain of the perception of an odor in connection with the nasal disease with which he is affected. Parosmia is due to involvement of the olfactory nerves.

A sense of *dryness* is a symptom of which the patient frequently complains, particularly in the early stages of acute rhinitis and throughout the entire course of a dry catarrh, or atrophic rhinitis.

Obstruction or Stenosis. This causes sometimes the greatest discomfort to the patient. There may be simply a sense of stuffiness and fullness in the nasal and frontal region, or complete obstruction, causing inability to breathe through the nose. It occurs in all the obstructive diseases of the nose and naso-pharynx; in acute rhinitis, in chronic inflammation (except the atrophic form), in hyperæmia, the hypertrophies, polyps, tumors, deviations of the septum, foreign bodies and adenoid vegetations.

Deafness is complained of when the Eustachian tubes are invaded or obstructed from inflammation or stenosis. When associated with anosmia it may be of central origin. *Tinnitus aurium* frequently accompanies the deafness.

Cough. This is of an irritative character. The discharge may pass into the pharynx and the larynx, setting up an irritation on account of which cough takes place. It occurs, therefore, in the catarrhs and obstructive diseases, and is not diagnostic of any nasal condition. When the nostrils are too wide, as in atrophic rhinitis, cough may occur because irritating particles are admitted through the widened aperture.

The Data Obtained by Observation.

THE OBJECTIVE SYMPTOMS. Of the general objective symptoms, fever has been noted. In certain affections of the nose defective development of the general system is observed. This is particularly the case in adenoid vegetations of the naso-pharynx in children. (See Diseases of the Pharynx.)

Local Examination. The Exterior. The external appearance of the nose is one of diagnostic significance when marked deformity takes place. Its true shape is changed in myxœdema (*q. v.*), but is not of consequence except in association with other symptoms of that affection. The change in the shape of the nose, of special significance, is seen in cases of disease of the bone due to syphilis. The bridge of the nose is sunken, or depressed. It must not be confounded with the depression that occurs in fracture. The nose may be broadened in cases of tumors of an expanding nature in the nasal cavities. The local change soon extends to the cheek. The nose also is the seat of eruptions, as acne and hyperæmia, but they are usually of local origin. The latter may be suggestive of a gouty diathesis.

Internal Examination. The examination of the cavities of the nose consists of two procedures, both of which are necessary to determine with accuracy the condition of the organ. These are:

1. *Anterior Rhinoscopy.* For this are needed a good light, a nose speculum of some form, probes, a 10 per cent. solution of cocaine, and a head mirror with central opening.

The examiner proceeds as follows: The patient is seated facing the surgeon, with the light behind and at one side of the head, as nearly as possible on a level with the eye of the operator. He must sit with shoulders and head a little forward. The operator adjusts his head mirror so that the central aperture is in front of his own eye and the reflected light falls on the nose of the patient. It is very important for nose examination that the operator look through the aperture and not *under* the mirror. The speculum is then taken in one hand and the nostril dilated so that the view of the interior is unobstructed. Do not try to dilate the bony part of the nose, but only the nostril. Proceed from before backward with the examination, carefully focussing the light on each part in succession, and gradually tilting the head of the patient backward. Thus the floor of the nose, the septum, inferior turbinated bones, middle turbinated bones, and sometimes the superior turbinated bones, are brought into view successively. In a broad nose one may at times see the posterior wall of the pharynx, which is distinguished by its peculiar wave-like movement when the patient swallows. The use of the probe is important, and without it no positive diagnosis can be made. With the probe the operator tries the condition of the mucous membrane, tests the consistency of tumors or hypertrophies, and so judges of the character of the condition. After this the enlarged parts should be touched with the cocaine and the result observed. Contraction of a swelling under its influence proves its vascular origin.

2. *Posterior Rhinoscopy.* This is the most difficult part of the examination, and requires much practice to enable the operator to accomplish it satisfactorily. The instruments needed are a tongue depressor, head reflector, two sizes of throat mirrors, a palate hook or flat strings for holding forward the soft palate, and a curved applicator for cocaine, or a spray bottle with tip turned upward.

The patient is seated as before, the tongue held down by the tongue depressor, and the patient is told to breathe freely through both mouth

FIG. 21.

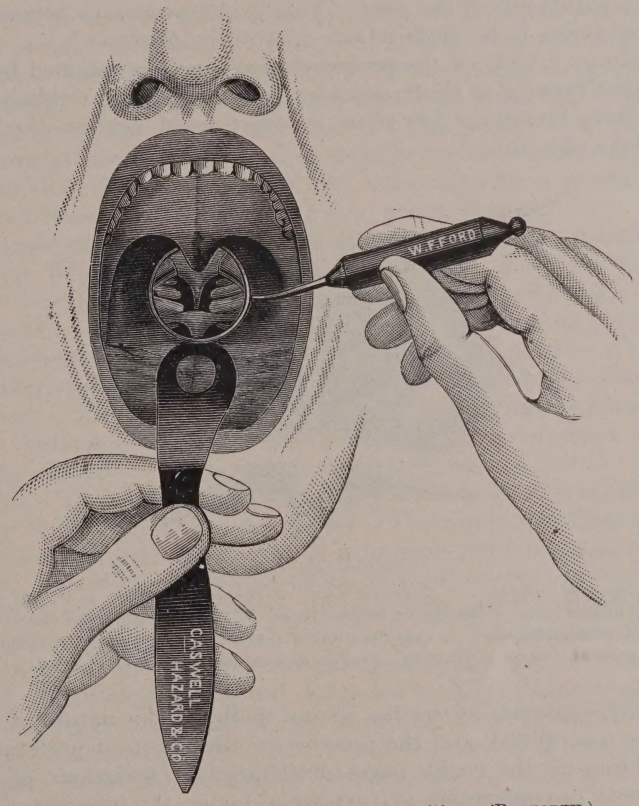


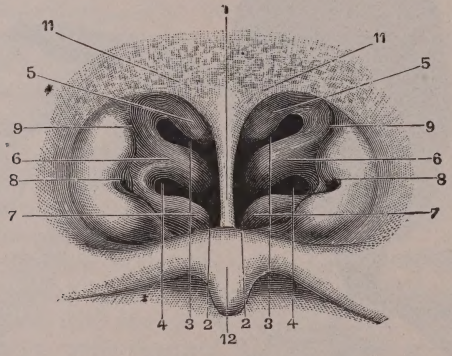
Diagram showing rhinoscopic mirror in position. (BOSWORTH.)

and nose. The light is directed into the pharynx and a mirror of the largest possible size inserted carefully behind the soft palate. The proper angle and the movement necessary to bring all parts into view can only be learned by practice. As a rule, it is best to hold the handle well up at first and note the condition of the vault of the pharynx, then gradually depress it, examining the choanæ from above downward. Do not keep the mirror too long in the throat. It is better to insert it several times than to weary the patient by attempting to see everything the first time. After the choanæ have been examined a turn of the mirror to either side will bring into view the orifices of the Eustachian tubes,

and the examination is complete. Where it is impossible to see the posterior nares after repeated attempts, one must first seek to accustom the patient to the presence of the instruments; if this fails it may be necessary to resort to the palate hook or the cords to hold the uvula forward. The best hook is White's. It is necessary to apply cocaine to the soft palate before inserting the hook. Another plan which is preferred by some is to take the flat cords used for corset laces, soak them in mucilage and dry them. These are then stiff enough to pass through the nostril, yet flexible enough to pull down and out through the mouth with forceps. Then by drawing forward both ends the soft palate is pulled out of the way. This is almost always necessary when applications are to be made to any spot in the pharynx.

Sometimes a view of the posterior nares may be obtained by making the patient breathe in short, quick gasps, by which the uvula is released. In ordinary breathing it is often tightly pressed against the posterior wall of the pharynx.

FIG. 22.



Rhinoscopic image.

1. Vomer or nasal septum. 2. Floor of nose. 3. Superior meatus. 4. Middle meatus. 5. Superior turbinated bone. 6. Middle turbinated bone. 7. Inferior turbinated bone. 8. Pharyngeal orifice of Eustachian tube. 9. Upper portion of Rosenmüller's groove. 11. Glandular tissue at anterior portion of vault of pharynx. 12. Posterior surface of velum. (SEILER.)

Through examination by the above methods the nature of the discharge is ascertained, and the presence of ulceration or perforation and the condition of the entire nares determined. Deviations of septum, enlargement or contraction of turbinated bones, the presence of foreign bodies or abnormal growths, are also ascertained in this manner.

Palpation. In palpation the finger or probe is used. By the latter the character of enlargements or tumors, and the patulency of foramina may be determined. The character of the mucous membrane as to induration and the presence of caries or necrosis is estimated. By the finger the naso-pharynx is palpated to confirm the results of rhinoscopy. In this manner adenoid vegetations and hypertrophy of the inferior turbinated bones are detected. The finger should be protected by the use of a mouth-gag or by a jointed thimble.

Color of the Mucous Membrane. The observer may find it unusually pale. This is seen in tuberculosis and in atrophic rhinitis. If a

protuberant mass is observed to be transparent and shining, as well as pale, it is due to a polypus. If the mucous membrane is bright red it may be due to acute inflammation, to glanders, or to syphilis. It is dull red in chronic catarrhs and caseous rhinitis. The coatings of the mucous membrane are of significance. If a dry mucus covers the part it is due to dry catarrh ; on the other hand, a dirty-gray membrane is indicative of diphtheritic rhinitis.

Ulceration of the Mucous Membrane. Ulceration is usually a manifestation of lupus, tuberculosis, or tertiary syphilis. In lupus the ulceration has extended from the exterior. Tuberculous ulcers are usually found in the septum. They present a whitish-gray surface with elevations of infiltrated tissue. They are liable to bleed on the slightest provocation. The mucous membrane surrounding them is torn. Tubercle bacilli can be found in the scrapings from the ulcer. In syphilis the ulcers are situated anywhere in the nares. They may be mere superficial excoriations, or deep serpiginous ulcers surrounded by an inflammatory zone. Caries may be detected with a probe. The ulcerated surfaces are covered with a dry, greenish crust.

Neuro-paralytic ulcers are painless, spread rapidly over considerable surface, and follow paralysis of the fifth nerve. They are dry and sluggish ; they do not extend to the skin. Post-febrile ulcers follow measles, scarlatina, typhoid, and variola, and are due to rupture of small abscesses, with the subsequent formation of ulcer. They are usually anterior on the septum or inside the alæ, and scabs form over the surface. They are very irritable. Ulcers may perforate the septum or the floor of the nose. They are usually due to syphilis. Simple perforating ulcer of neuro-paralytic origin may also occur.

Secretion. The study of the secretions is of diagnostic significance. They may be liquid, semi-solid, or solid. The liquid secretions may be serous, mucous, or purulent. *Serous* secretions occur in acute rhinitis, hay fever, and idiopathic rhinorrhœa, and follow bursting of cysts. The secretion of *mucus* occurs in the later stages of inflammation of the mucous membrane and in chronic forms. A *muco-purulent* secretion is seen in chronic rhinitis, and pure *pus* in abscesses of the septum or cavity. A discharge of blood is known as epistaxis (see page 194). The *semi-solid* secretions may be due to mucus alone, or to blood-clots mingled with serum or with pus. The latter occur in atrophic and hypertrophic catarrhs. A semi-solid secretion is seen in Caseous Rhinitis. On examination the cavities are filled with cheesy matters, easily broken up with the probe. The mucous membrane is dull red. The material is discharged in masses at intervals through the mouth or nostrils, relieving the previous extreme stenosis. If neglected for a long time, deformity of the face and disease of the bones and cartilages of the face ensues from pressure.

The *solid* secretions may be mucous crusts, as in acute and chronic catarrhs, blood crusts after epistaxis and traumatism, membrane in diphtheritic rhinitis, sloughs from ulcers, and rhinoliths.

Microscopical Examination of the Nasal Secretion. The normal secretion from the nose contains squamous and ciliated epithelium, isolated leucocytes, and various fungi. The fluid is thick, alkaline in reaction,

of slight odor. It contains mucin. In disease of the nasal cavities the fluid changes. In acute nasal catarrh it is more copious and thinner. It remains alkaline, and contains epithelium and fungi. When the stage of suppuration is reached, pus may almost entirely compose the secretion. Cerebro-spinal fluid may also be discharged through the nose in certain brain tumors. In such fluid albumin is absent. Detection of this fluid is of diagnostic value, pointing to the central lesion.

In diphtheria the characteristic micro-organism is seen. Recognition of glanders may be based upon finding the bacillus in the nasal secretion (see Blood). Cultivations may be made. The nature of ulcers may be determined by microscopical examination. The tubercle bacillus can be detected at times. A pneumococcus has been found, or bodies that resemble it, in the secretion in ozæna. Thrush fungi have also been found, as well as some mould fungi. The Charcot-Leyden crystals are found in the nasal secretion in asthmatic patients, sometimes in acute coryza.

Mouth-breathing. Much valuable information is obtained by noting the character of the breathing and the condition of the voice. Mouth-breathing is liable to be present if the face is drawn and vacant and there are cracks and fissures in the mouth. With mouth-breathing the voice is usually nasal. The resonating quality is lost entirely. Snoring accompanies these conditions, and they are all due to obstruction of the nares. (See Obstructive Symptoms.)

Epistaxis. The blood may flow in drops, or a continuous stream pour out from the anterior nares. Sometimes it falls into the pharynx and is hawked up, or is swallowed and then vomited.

It may occur from local causes, or be the result of constitutional conditions. Traumatisms (scratching the nose) new growths, and foreign bodies are causative agents. It may be due to fractured skull. Local causes: On inspection, the cause may be found in enlarged veins at the anterior inferior portion of the septum, a bleeding ulcer, a new growth, or the ulceration of a foreign body. The general conditions which are causal are: (1) Plethora; (2) engorgement due to the rising of an elevation; (3) all forms of anæmia; (4) it is the common seat of bleeding in hæmophilia; (5) cerebral congestion and severe headache; (6) in the commencement of fevers, and particularly typhoid fever, it frequently takes place. In children exposed to the sun, and after exertion, it is of frequent occurrence, and is seen often at puberty in delicate children.

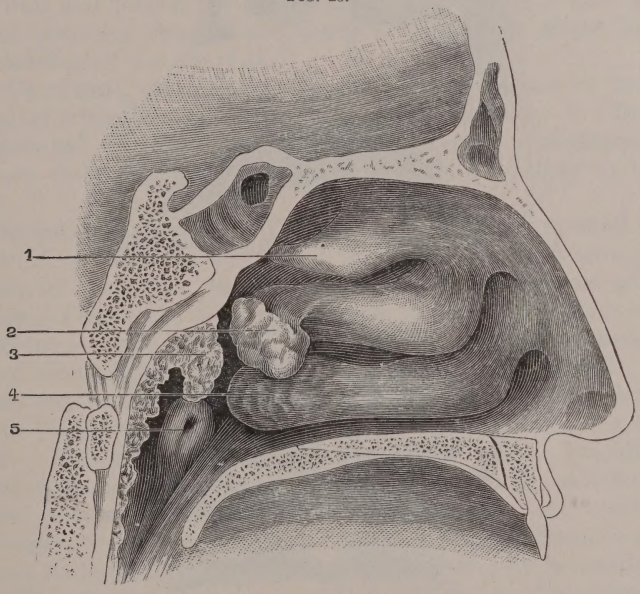
Disease of the Nose.

Catarrhs of the Nose. These may be acute or chronic.

Simple Acute Rhinitis. *Acute Coryza, "Cold in the Head."* Beginning with a feeling of lassitude, aching in the back and limbs and feverishness, a sense of fulness is felt in the nostrils, with sneezing. After twenty-four hours an irritating discharge from the nostrils begins. During this time the malaise has increased. The pain in the forehead and cheeks has become more pronounced, and a nasal twang is given to the voice. The feverishness continues, reaching 101° in the more pronounced cases, with thirst and loss of appetite. At the height of the fever, in forty-eight hours, very often a crop of herpes develops on the

lips. The general symptoms then subside and the local symptoms change. The discharge becomes thick and purulent, the fulness continues, but the pain is diminished. The inflammation has extended up the tear-ducts and to the eyelids. These are congested and may burn and be irritated. Very frequently, also, the inflammation extends to the pharynx, causing soreness of the throat and stiffness of the neck, and the larynx even may be involved. A slight deafness may arise from the inflammation extending into the Eustachian tube Rhinoscopic examination of the mucous membrane shows it to be red and swollen during the first day. The discharge, as described above, is secreted from it. The contractile tissue over the turbinated bones is congested and swollen, on account of which the nasal passages are occluded. To the probe the tissue is elastic, and it contracts promptly when cocaine is applied. The coryza may be symptomatic of measles, hay fever, or influenza.

FIG. 23.



Vertical section through nasal cavities. (Diagrammatic.)

1. Superior turbinated bone. 2. Middle turbinated bone, with posterior hypertrophy. 3. Section of hypertrophied pharyngeal tonsil. 4. Inferior turbinated bone. 5. Orifice of Eustachian tube. (SEILER.)

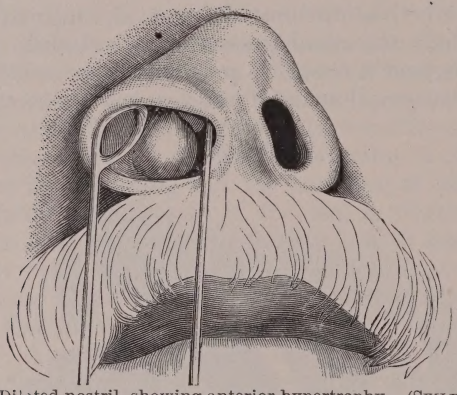
In the DIPHTHERITIC FORM of acute rhinitis the diagnostic symptom is the presence of the false membrane in the nose. If, during the presence of diphtheria, a sloughing coryza occurs and the cervical glands are found to be swollen, careful examination of the nose should be made. The discharge is very acrid in diphtheria, and is almost sure to cause excoriation of the upper lip, the presence of which condition under the above circumstances is of great significance. On rhinoscopic examination, a dirty-gray membrane is found lining the nostril. Bacteriological examination confirms the diagnosis.

CHRONIC RHINITIS. Four varieties may be distinguished, to all of

which the term *nasal catarrh* may be applied. In one there is hypertrophy of the turbinated bones; in the second, there is extension of the disease to the post-pharynx—chronic post-nasal catarrh; in the third there is absolute dryness of the mucous membrane—rhinitis sicca, or dry catarrh; in the fourth there is atrophy of the mucous membrane—atrophic rhinitis, or ozæna.

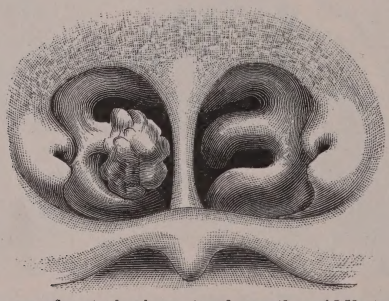
In CHRONIC HYPERTROPHIC RHINITIS, the affection comes on gradually after repeated acute attacks. The only symptoms may be

FIG. 24.



Dilated nostril, showing anterior hypertrophy. (SEILER.)

FIG. 25.



Rhinoscopic image from a case of posterior hypertrophy on the middle turbinated bone. (SEILER.)

slight fulness in the nose and a little hoarseness of the voice. In more advanced stages, the symptoms of stenosis are marked with oral breathing, snoring, and nasal sound. From the nostrils a constant discharge of muco-pus takes place, which is discharged backward into the pharynx, causing hawking. The hearing is frequently impaired, as well as the taste and smell. The discharge often affects the larynx, causing an irritating cough. The hypertrophied tissue on the turbinated bones, or pressure of the bone on the septum, may lead to reflex attacks of asthma.

Rhinoscopic Examination. The uvula is thickened and elongated on account of the hawking. The outer surface or the edges of the turbinated bones are enlarged, either generally or in places. The mucous mem-

brane covering these spots is thickened, hard, and rough. If cocaine is applied, the mucous membrane does not contract, as in the swelling due to hyperæmia. The posterior ends of the inferior or middle turbinated bones are enormously enlarged, forming round tumors which obstruct more or less the posterior nares and project into the pharynx; polyps and deviation of the septum complicate these cases.

Chronic Post-nasal Catarrh is an extension of the former into the pharynx. It is distinguished by discomfort or pain in the soft palate and posterior nares. There is tingling and a sense of fulness at the root of the nose, frontal headache is present, the patient complains of a bad taste in the back of the mouth and of constant flow of thick secretion into the pharynx, causing snoring and hawking. The same perversions of the sense of taste, smell, hearing, and of the voice occur as in acute rhinitis. In *rhinoscopic* examination, in addition to the appearances in the nares, there is a mammillated appearance of the anterior wall and floor of the pharynx, with thickening of mucous membrane and posterior third of the septum. Headache seems to be due to the condition of the pharynx.

Dry Catarrh, or Rhinitis Sicca, is also chronic in its course, accompanied with tingling and dryness of the nostrils. A faint, musty odor is detected, but there is no discharge or sense of obstruction. In severe cases there may be sharp pain in the nose extending to the forehead.

Rhinoscopic Examination. The mucous membrane is coated with dry mucus, while crusts form constantly, giving rise to much annoyance.

Atrophic Rhinitis, or Ozæna, is attended by a sense of dryness in the nose. Occasional obstruction arises from accumulations of crusts, otherwise the passage is unduly open. There is constant hawking and spitting of brownish-green crusts which are often blood-tinged. Frontal headaches may occur in paroxysms. The spirits of the patient are often depressed. The *odor* is characteristic, and is diagnostic if syphilis is excluded. The bridge of the nose may fall in slightly. On *rhinoscopic* examination the mucous membrane is found to be thin, pale, hard to the touch, and covered with a layer of dried secretions and crusts. The nasal passages are abnormally wide and the turbinated bones very small. There may be hypertrophy in one nostril and atrophy in another.

In addition to the above, a so-called Strumous Rhinorrhœa is seen in scrofulous children. There is a continuous discharge of mucopus from the nostrils, which are obstructed by the swollen mucous membrane, and particularly by greenish-yellow crusts.

Syphilitic Coryza is seen in infants and young children affected with hereditary syphilis. The discharge is at first thin and mucopurulent. It soon becomes thicker and more purulent, later thin and sanious. The nostrils are swollen and red at the edges, sometimes completely occluded, causing oral respiration and inability to take the breast or bottle.

Pustules, fissures, and ulcers are found in the nose and at the margin of the orifices. They are also seen in the pharynx and larynx. Hemorrhages may occur. Other evidences of hereditary syphilis are present.

Rhinitis Caseosa has been described previously (see Secretion).

Nasal Polypi.

On account of the presence of polyps in the nostrils there are symptoms of stenosis. A sense of fulness and obstruction attended by oral breathing and snoring is common. An acute rhinitis or damp weather aggravates the symptoms. If neglected, conjunctivitis arises, on account of pressure on the lacrymal ducts. Epistaxis and sneezing are of frequent occurrence.

Rhinoscopic Examination. The polypus is seen as a grayish-yellow or greenish *shining* mass projecting by a broad base from the mucous membrane. The probe shows that it is soft and yielding and that it can be circumscribed.

Foreign Bodies.

Animal parasites may find their way into the nostrils and act as foreign bodies, or substances may be thrust into the nostril. There is stenosis and secondary ulcerative rhinitis with fœtid sanious discharge, often purulent.

Rhinoscopic Examination. The foreign body may be seen at once or an ulcer only with granulating edges, be detected. The body is in the ulcer; the probe, which must be used thoroughly, can usually detect it. Only in the tropical regions, usually, are parasites found in the nostrils. They are the larvæ of the *lucilia hominivora.* It is said that the pain is so severe at the root of the nose, and thence extending backward, as to cause maniacal delirium. Sleeplessness is present, and there may be extensive destruction of the bones and skin. There is a fœtid sanious discharge. Simple vegetable or inorganic bodies, as peas, beans, buttons, hair-pins, etc., cause pain which may become intense if the body is of vegetable origin and swells.

Rhinoliths are foreign bodies in one sense, and yet they develop in the nostrils. They are gray or greenish-brown in color, hard and rough, either fixed or movable. They sometimes cause pain and reflex neuroses.

Nasal Tumors.

Tumors of the nose other than polypi partake of the same characteristics as tumors in other situations, and lead to symptoms of obstruction with internal and external deformity. In the beginning practically the symptoms are similar to those caused by a foreign body. Fibroma, sarcoma, osteoma, and enchondroma are seen. Malignant polypi or carcinomata grow rapidly. They extend over a large surface and are attended by pain. They bleed easily and cause a fœtid, sanious, ichorous discharge. Epistaxis is common. Stenosis and deformity are marked. The glands of the neck are swollen.

Glanders.

This rare disease affects persons in contact with horses that have it. General symptoms consisting of pain in the trunk and limbs, with rigors followed by fever, occur first. Nausea and vomiting and diar-

rhœa attend the first twenty-four hours of the attack. There may be dyspnœa. A typhoid type of fever is present. A pimple appears on the skin, which becomes painful and swollen, and at the same time a thick, yellowish discharge streaked with blood oozes from the nostrils. Hard pustules appear around the nose and in other parts of the body. Death occurs from exhaustion. (See Glanders, and The Blood.)

Ulcerative Diseases of the Nose.

We have to distinguish the *syphilitic* and *tuberculous* ulcer and the ulcer of *lupus*. In the former a history of infection, or of secondary and tertiary manifestations, can be obtained. The stench of the breath is sickening, and the patient complains of stenosis and loss of smell. There is some localized tenderness, and sleeplessness, debility, and emaciation may ensue. In tuberculosis, ulcers tend to bleed readily. They are usually secondary to tuberculosis in some other region of the respiratory tract. Microscopic examination of the scrapings from the ulcer reveal tubercle bacilli.

If ozæna is present in a patient in whom lupus is seen on some part of the external surface, there is also probable lupus of the nasal passages. The ulcers may be followed by necrosis and caries of the bones. If the ozæna is not removable by antiseptic sprays the bones are probably affected. A discharge of sequestra makes the diagnosis positive. Rhinoscopy and careful palpation may reveal the ulcer and a carious bone.

The Auxiliary Cavities of the Nose.

The Antrum is subject to abscess, cysts and polypi, parasites, and tumors.

Abscess. An odor somewhat like that of ozæna, a putrid taste, nausea, anorexia, pain in the cheek and root of the nose, often neuralgia in the frontal region, and malaise, are present. A very characteristic symptom is the discharge of pus from one nostril on leaning the head forward. There is often a bad tooth on the same side in the upper jaw.

The Sinuses. The frontal, ethmoidal and sphenoidal sinuses are subject to inflammation, abscess, traumatism, and the irritation of foreign bodies, usually parasites.

The frontal sinuses are the only ones which exhibit external symptoms. When these cavities are inflamed there are pain and tenderness over the frontal protuberances; if the process goes on to the formation of abscess there may be redness and swelling and finally fluctuation. If the communication is not closed there is a fœtid discharge from the middle meatus.

When the sphenoidal and ethmoidal sinuses are affected there are no external symptoms unless the enlargement is so great as to affect the orbit. There is deep-seated pain. Pus is seen exuding into the superior meatus and flowing backward into the pharynx. Parasites cause intense pain and lead to abscess, caries, and necrosis. *Rhinoscopic* examination shows in disease of the antrum rough hypertrophic enlargement on the under surface of the middle turbinated bone and a flow

of pus into the middle meatus. Sometimes a probe can be passed into the antrum from the nose. Often an exploratory puncture is necessary. *When the foramen is obstructed* there is a dull aching pain in the upper jaw, with deformity of the orbit, face, hard palate, and nostril. Fluctuation can usually be found at some point after a time.

The *lacrymal duct* and sac are often the seat of inflammation by extension, on account of which there is pain, some obstruction in the nose, and epiphora. On examination pus will be seen flowing forward over the inferior meatus. By the lacrymal probe the ducts are found to be painful and obstructed, and pus exudes.

Reflex Neuroses.

Bronchial Asthma. Asthma may be due to disease of the nose, but the only proof that it is of nasal origin is in its disappearance after treatment of the various faults in the nose, on account of which it may have developed. *Hay fever* is an acute affection ushered in by paroxysmal sneezing, itching, and smarting of the inner canthus of each eye, or of the throat or nose. After hours or days of sneezing coryza develops. The disease continues for a varying length of time, is more pronounced at certain seasons of the year, particularly the late fall. Coughing may be an additional symptom, and paroxysms of asthma may develop which are hard to distinguish from true bronchial asthma. The attack may be excited by emanations from vegetation, particularly the pollen of plants, but other emanations may also induce it. The condition of the nasal mucous membrane predisposes to the attack. Local inflammation of the nose or obstructive disease from hypertrophies is primarily present. To the exciting cause and the local predisposing cause may also be added a neurotic factor. The disease affects families of nervous constitution, and may occur through several generations. It is more common in this country than in other countries, and dwellers in cities are more subject to it than residents in the country.

Idiopathic Rhinorrhœa. Characterized by a sudden profuse discharge of yellowish water. It ceases as suddenly as it develops, and is thought to be due to some functional derangement of the fifth nerve.

Diseases of the Larynx.

The structural composition of the larynx does not differ from that of other parts of the respiratory passage. Mucous membrane, connective tissue, cartilages, and muscle are similar to these tissues elsewhere situated.

The result of their anatomical association in the larynx is the establishment of the functions of that organ, the formation of the voice and the passage of air into and out of the trachea. Now, the morbid processes that affect the larynx do not differ from the morbid processes elsewhere in which similar tissues are involved. Each tissue is liable to congestion, to inflammation, to degeneration, to new-growth formation. The joints may become ankylosed, the muscles either paralyzed or the seat of spasm. The symptoms common to morbid processes in each class of tissue are seen. But other symptoms arise because of the

anatomical position of the larynx and of its functions. The narrow chink of the glottis soon becomes occluded, and hence *dyspnœa* arises. Obstruction to the pathway or pain from inflammation or ulceration causes *dysphagia*. The sensitiveness of the mucous membrane provokes *cough* on the slightest provocation. The cords cannot vibrate or muscles and articulations cannot move, and *dysphonia* or *aphonia* occurs.

The larynx is a highly specialized organ, and is well innervated. Large central nuclei, connected by a large nerve which passes over a circuitous route and which anastomoses with other nerve cords, preside over the function of phonation. Affections of the central nuclei, affections of the nerve trunk or of the structures adjacent, thereby pressing upon the trunk, have their expression in disorder of the larynx, and particularly if with disturbance of phonation. In other words, the phenomena of laryngeal disease may be symptomatic of disease of the brain or of the nerve trunk, as well as disease of the larynx. (See Nervous Diseases.)

Because of its anatomical position and special function the symptoms of disease of the larynx are very striking, calling attention at once to their seat. Laryngeal affections are not liable to be mistaken for disease of contiguous parts, although retro-pharyngeal abscess, abscess at the side of the pharynx, disease of the thyroid gland, and inflammation of the lymphatics or cellular tissue in the neck may cause symptoms suggestive of laryngeal disease.

Finally, morbid processes in the larynx determined by the symptoms and physical appearances may be symptomatic of general processes: acute inflammation, of erysipelas, typhoid fever, smallpox, or measles; chronic inflammation or ulceration, of the rheumatic or gouty diathesis, syphilis, or tuberculosis; scars, of syphilis; ankylosis, of rheumatic gout. The laryngeal symptoms of brain disease or of affections of the nerve trunk have been referred to.

The practical point of all this is that affections of the larynx are not due to primary disease of that organ alone, but are often secondary to general processes or local morbid processes elsewhere.

Therefore, when laryngeal symptoms or lesions are observed, seek beyond the larynx as well as in it, for their cause.

The Data Obtained by Inquiry.

Subjective Symptoms. *Pain.* Pain in the larynx may be sharp, stabbing in character, or simply a tickling or burning with a feeling of pressure. Pain is sometimes so intense as to render speaking and swallowing impossible. In acute laryngitis the pain is cutting and burning. In the milder inflammations, in dry catarrh, and in lupus it amounts to soreness only. The pain is severe and sharp in cases of cancer and tuberculosis, rarely in syphilis, and when foreign bodies are present in the structures. The pain may be very severe and intense when there is destructive ulceration. It is a diagnostic symptom of perichondritis. Usually the pain is localized in the larynx, but in ulceration it may extend to the ears. This is particularly true in carcinoma. The pain is propagated by the auricular branches of the vagus.

Pain is increased by pressure in all affections of the larynx, and intensified by the act of swallowing and by speaking.

Paræsthesia. Peculiar sensations are frequently complained of. They may be *burning*, *tickling*, or *itching* in character, or it may seem as if a foreign body were present in the part, as a hair, or it may seem like a draught of cold air striking the parts. Sometimes after a foreign body has actually been present, the sensation of its presence will continue a long while after its removal. A sense of pressure or fulness, the feeling of a lump in the throat, is frequently complained of, provoking a desire to swallow. The patient will seek advice on account of it. It is known as the *globus hystericus*, and is recognized by the absence of local changes in the larynx, by its association with other phenomena of hysteria, and by its disappearance or aggravation under the influence of excitement. This abnormal sensation is seen in hysteria and hypochondriasis. It is one of the nerve perturbations in chlorosis and anæmia.

A feeling of *dryness* is frequently complained of, and attends the acute stage of acute laryngitis, and chronic laryngitis. The sense of fulness, or pressure, or feeling of the presence of a foreign body is complained of in all forms of laryngitis, in croup, in œdema of the glottis or epiglottis, and in syphilitic infiltration.

Hyperæsthesia and Anæsthesia. When there is *hyperæsthesia* there is constant desire to *cough* (see page 204), and the act is aroused on the slightest irritation. The desire to cough, independently of the act, however, is of itself an extreme annoyance. It is a disagreeable sensation present in acute inflammations and in early phthisis. At times of menstruation and during pregnancy both symptoms are frequently complained of. Cough occurs reflexly in dentition. Hyperæsthesia is easily recognized with the probe. In *anæsthesia* particles of food fall into the larynx. The mucous membrane is insensitive to the contact of the sound. Anæsthesia occurs in hysteria, diphtheritic paralysis, paralysis of the superior laryngeal nerve, bulbar paralysis and cerebral softening or hemorrhage, or coma from any cause.

Mis-swallowing, or "swallowing the wrong way," occurs in all conditions in which food is allowed to enter the larynx. Although conditions favorable for its occurrence are present it may not take place unless the patient is off guard during the act of swallowing, as when laughing is provoked. It may then occur even in normal cases. It is associated with anæsthesia of the larynx, and occurs in central nerve affections which cause that condition.

Dyspnœa. This is one of the frequent symptoms—and the most serious—of laryngeal disease. It occurs when obstruction takes place, and may be due to spasm, to inflammatory or œdematous swelling of the tissues of and about the larynx, to tumors or foreign bodies in the larynx, to the cicatrization of ulcers after syphilis or lupus, to paralysis of the abductors or adductors of the larynx. Disease of surrounding structures which press upon the larynx causes dyspnœa, which is similar to that due to actual disease of the organ.

Dyspnœa may vary in degree from slight inconvenience in breathing, only felt by the patient, to the violent struggling for breath which is

seen in cases of extreme stenosis of the larynx. If carefully observed in either case the larynx is seen to rise and fall. If the obstruction is present in its more aggravated form the head is bent back, the neck stretched, the muscles of the neck contracted. The spaces above the sternum and at the sides of the trachea are drawn in with inspiration, and the alæ of the nose work vigorously. Further evidence that sufficient air does not enter the lungs is seen in the recession of the epigastrium and the drawing in of the ribs at the base of the chest during the act of inspiration. At the same time the countenance is dusky or ashy-gray, the lips become cyanosed and the nails bluish as the dyspnœa persists and deepens. A cold perspiration breaks out on the forehead, and finally, from exhaustion, the respiration becomes slower and slower until mere gasps are seen. The heart's action increases in frequency as the stenosis increases. Death usually takes place from asphyxia, the child first falling into a stupor on account of carbonic acid poisoning. The dyspnœa under these circumstances in the various degrees described is generally *inspiratory*. Noise attends the act of inspiration, the character of the sound depending on the nature of the obstruction. If the obstruction arises from simple spasm, or from intense inflammation of the larynx, without secretion, the sound of inspiration is harsh and stridulous. In obstruction that occurs from œdema or from exudation, as in laryngeal diphtheria, the sound of inspiration is loud and stridulous but not shrill. The expiration is usually noiseless and prolonged. The short, stridulous, or gasping inspiration is followed by prolonged gentle expiration. In spasmodic croup, the expiration is like snoring. The interval between expiration and inspiration is lessened, the respirations are hurried.

In another form of dyspnœa the obstruction takes place when the air is passing out of the lungs, as in cases of a movable tumor below the vocal cords. The act of inspiration is complete, the act of expiration is suddenly checked by the obstruction, on account of which the lungs become overfilled with air, and an emphysema develops. In another variety, laryngismus stridulus, the act of breathing ceases in the midst of inspiration. Cyanosis develops (see Color, page 72).

The dyspnœa from disease of the larynx may develop gradually and continue over a long period of time, or it may be acute in onset, depending upon the character of the morbid process on account of which the obstruction has taken place. Acute paroxysms of dyspnœa, in one of which a fatal ending may take place, are liable to occur in the course of affections in which chronic dyspnœa is present; thus sudden œdema may occur in cases of syphilitic or tuberculous ulceration.

Laryngeal dyspnœa must be distinguished from other forms of dyspnœa. This has been considered elsewhere with regard to the dyspnœa due to diseases of the heart and lungs. 1. The dyspnœa that occurs on account of pressure upon the trachea differs. The larynx is not markedly moved during the respiratory acts, and the patient bends the head forward instead of backward. 2. The diseases which cause dyspnœa from pressure on the larynx must be excluded. Cellulitis of the neck, tumors of the lymph glands, goitre and retro-pharyngeal abscess are provocative of this form of laryngeal dyspnœa. Examination of the

respective localities by inspection and by touch reveals the cause. It may be worthy of remark that dyspnœa in diphtheria, frequently thought to be due to internal occlusion, may be due to pressure of enlarged glands on the bronchus and larynx.

Dysphagia. Difficulty of swallowing is most marked when destruction of tissue in the larynx takes place, or when there is acute inflammation about the muscles or their attachments; hence, when ulcers, tuberculous or malignant, are present, or perichondritis arises, the difficulty is so great with the pain that arises as to prevent the taking of food. When the epiglottis is the seat of acute inflammation there is great dysphagia on account of pain, or perhaps on account of the obstruction. When the epiglottis is fixed, and in forms of ulceration of the larynx, particularly if that structure is involved, the food enters the larynx, and hence dysphagia is produced.

Dysphagia is recognized by pain and by the falling of particles of food into the larynx, exciting cough. It must be distinguished from the dysphagia of pharyngeal affections by ocular examination, the location of pain, and the non-association of rheumatism.

Dysphonia. The most common symptom of affections of the larynx is disturbance of the function of speech. The voice is changed in character, or may be lost in any affection which causes swelling of the mucous membrane, or occlusion of the orifice, or which interferes with the action of the vocal cords. The voice may be hoarse in acute and chronic inflammations, in tumors and in specific ulcerations about the larynx, and in paralyses of the cords. From simple hoarseness it may vary in intensity to complete aphonia. The laryngoscopic examination is necessary in order to detect the presence or absence of paralyses. (See Paralyses.)

The character of the voice may change. When one-sided paralysis of a cord is present the voice is flat and toneless. In cases of paresis of the tensors of the cords a falsetto voice is created. Diplophonia occurs in one-sided paralysis, and in some cases in which small tumors lying between the cords come up during the act of phonation and form nodes. Two tones are formed at the same time in this class of cases. Frequently only certain tones are doubled. The duration may be significant. Hoarseness of long duration (years) is said to be prodromal of cancer. (Ziemssen.)

Functional dysphonia or aphonia may occur after excessive use of the voice and in hysteria. Hysterical aphonia occurs in women and young girls; the laryngoscope reveals nothing; the acts of coughing and sneezing are normal, and a sound may be created in either; it occurs or disappears suddenly.

Cough. (See Diseases of the Lungs.) Sometimes valuable information is derived from the character and severity of the cough. Several forms are noted:

First, the dry cough, as seen in acute laryngitis. It is almost constant, and is aggravated when the patient speaks, takes fluid, or inspires deeply. In children it is abrupt, brassy or metallic, stridulous or whistling, so-called "croup cough," as seen in cases of "false croup" and laryngitis with œdema.

Second, a dry hoarse cough occurs in the course of chronic laryngitis.

Third, cough with whoop. With the act of coughing a whooping sound may be heard in inspiration. After rapid violent expiratory acts with inspiration, the whoop takes place. It is spasmodic and convulsive, and is followed by retching, and often by vomiting.

Fourth, the cough is of such a character as to give one the idea that it is suppressed, in membranous and œdematous laryngitis.

Fifth, a cough frequently occurs without any local anatomical changes in the larynx, which seems to be purely of nervous origin. Two forms are seen: *a. Paroxysmal form.* Severe coughing occurs suddenly, and cannot be controlled by the patient. It ceases without cause, returning in a few hours. There is no expectoration. *b.* It may be *continued and rhythmical* in character. It is not so severe as in the paroxysmal form, but consists in a regularly recurring cough more or less loud. It does not occur while eating or speaking and ceases entirely during sleep. It is usually worse when the patient is under observation. Examination with the laryngoscope reveals absence of disease. This form of cough is seen after diphtheria, when sexual disturbances are present, at puberty, in cases of anæmia and chlorosis, or of neurasthenia or hysteria. The tone is usually high.

Hemorrhages. Hard coughing or an unusual straining of the voice may lead to the occurrence of slight hemorrhage. Only after injuries are hemorrhages from the larynx at all copious. Moderate hemorrhages occur in scurvy, hæmophilia, hemorrhagic smallpox, typhus fever, and leukæmia.

Disturbance of Co-ordination. Several forms of such disturbance are seen. Spasm of the glottis may occur with each effort to speak, causing either serious interference or complete inability to utter a word, as in stuttering. Sometimes, instead of the glottis opening to complete the act of inspiration, it may close. Sudden inspiratory dyspnœa, therefore, occurs, and is attended with stridor.

General Symptoms. In the study of laryngeal affections it is well to note objective phenomena distant from the organ or of a general character.

1. Fever, present in acute laryngitis and tuberculous ulceration. It is high in acute laryngitis with stenosis; in tuberculosis it is of a hectic type.
2. Cyanosis or cyanosis and pallor in laryngeal stenosis.
3. Extended and dilating alæ nasi in severe stenosis. Recession at the sternal notch and above the clavicles, and at the base of the thorax.
4. Cold sweating, sudden, with pallor, in laryngeal obstruction, as laryngismus stridulus, or when a foreign body is present.

The Data Obtained by Observation.

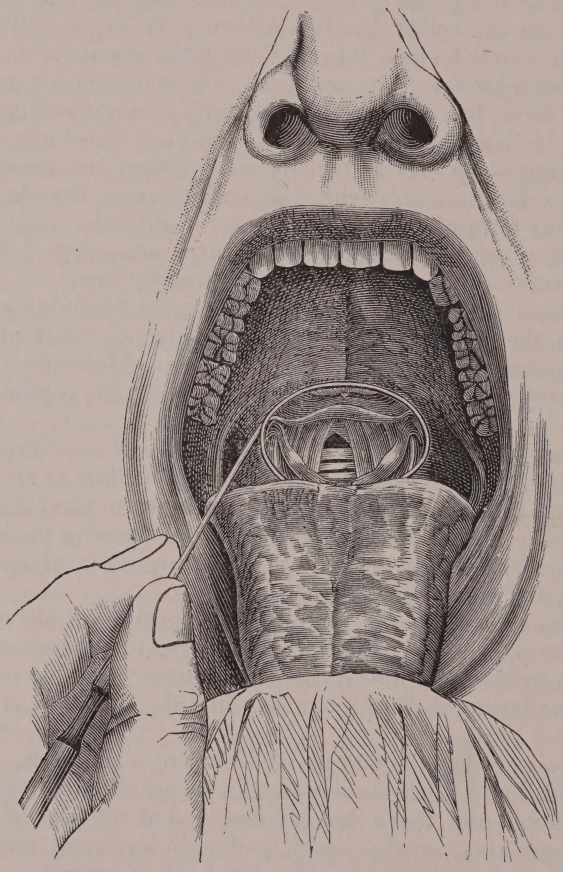
Objective Symptoms. The objective symptoms are determined by *inspection* and *palpation*. Inspection of the *exterior* of the larynx reveals the presence of swelling, and the movements of the organ as a whole. Local swelling of the tissues over the larynx may occur in inflammations of the cartilages; they are usually of syphilitic origin, but may attend carcinoma or follow tumor. There is more or less

marked swelling in inflammation of the cartilages, which after a time fluctuates, and when opened discharges pus and necrosed cartilage. The objective signs of inflammation are noted.

The movement of the larynx is increased in cases of dyspnœa. It is accompanied by recession of the spaces above the sternum and the clavicles, with clonic contraction of the sterno-cleido-mastoid muscle.

The interior of the larynx is studied by inspection (laryngoscopy), and by palpation (probe or fingers).

FIG. 26.



Laryngeal mirror in position, displaying the laryngeal image. (COHEN.)

LARYNGOSCOPY. In order to conduct laryngoscopy it is necessary first to have a good light. This may be direct sunlight, a good student's-lamp, or an Argand gas-burner. Electricity is not satisfactory. Second, a good reflector is required. It may be attached to a head-band or a spectacle-frame. It should be concave for artificial light, plain for sunlight, and should be pierced in the centre. Third, laryngeal mirrors of different sizes and a curved probe complete the instruments necessary for examination of the larynx.

Examination. The patient is seated with the source of light at one side and behind him; the head and shoulders are brought well forward and the head slightly raised. The operator takes a seat in front of the patient at a proper distance for the focal length of the reflector, and focusses the light on the patient's mouth, warms the laryngeal mirror over the flame and tests its temperature on the back of the hand. It should be raised to a moderate warmth, in order that when placed in the mouth the vapor of the breath may not precipitate on its surface. The patient must open the mouth and protrude the tongue, which is grasped between the folds of a napkin by the thumb and fingers of the operator. The tongue should be gently but firmly grasped. The mirror is then inserted carefully and quickly, face downward, into the pharynx. Care must be exercised not to touch the tongue or palate, otherwise the patient may be made to retch and he becomes alarmed. The mirror is passed to the posterior wall of the pharynx, and so directed that the image of the larynx is reflected to the eye of the operator. The patient is made to phonate "ā" or "ee," not "ah," and then to respire. The various structures and the action of the cords are observed. The appearances of the mucous membrane are studied during quiet respiration.

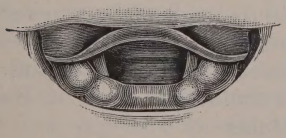
The epiglottis is very dependent, so that often the larynx can only be seen by having the patient stand while the operator remains seated. The patient's head is bowed on his chest and the examination proceeds.

The first examination may not result satisfactorily, but little being observed on account of the spasm of the pharyngeal muscles. Repeated sittings may remove apprehension and accustom the mucous membrane to the presence of the instrument. This object may be attained by administering the bromides, or by applying cocaine to the pharynx.

The probe is needed only to ascertain the consistency of tumors and growths. Cocaine must be applied before it is used.

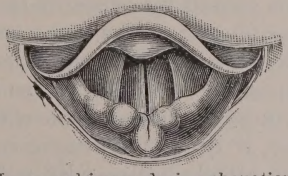
Sputum. The sputum from the larynx is generally scanty; it is not frothy, and is colorless and transparent; it is often discharged in small globules; it may be streaked with blood. Sometimes pseudo-membranes are coughed up. It is doubtful if purulent sputum ever comes from the larynx, excepting in cases of perichondritis in which the abscess bursts into the larynx. Laryngeal sputum is found in catarrhs and malignant tumors. It is blood-streaked when the catarrh is very intense, or after injuries.

FIG. 27.



Laryngeal image during respiration.

FIG. 28.



Laryngeal image during phonation.

APPEARANCE OF THE LARYNX IN HEALTH. Fig. 26 shows the larynx as it is seen in the laryngoscopic mirror. Above (upper part) is the arched epiglottis, below it the cavity of the larynx. In the centre are the vocal cords, white and glistening; on each side of

these the pink folds of the false cords. At the bottom of the mirror are the arytenoid bodies, and between them the folds of the inter-arytenoid space. Below and outside of the arytenoid bodies are the fossæ. The mucous membrane is pink throughout except on the cords. In respiration the arytenoids separate, carrying the ends of the cords which are attached to them with them, and leaving a triangular opening—the glottis—through which the rings of the trachea can be seen. (Fig. 27). In phonation the arytenoids approach each other, obliterating the inter-arytenoid space; the inner edges of the cords come in contact and close the glottis. (Fig. 28.) The appearances in disease are described under the different diseases. A note must be made of the color of the various parts, of the presence or absence of swelling or ulceration, and of the movements of the parts concerned in phonation. The latter particularly applies to the movements of the cartilage and of the cords, a full discussion of which will be found under Laryngeal Paralyses. Two conditions seen by the laryngoscope, common to many laryngeal disorders, will be spoken of in this place:

Anæmia of the larnyx may be merely a part of a general anæmia from any cause. In chlorosis it is seen before the external appearance is marked. An intense anæmia of the larynx is an early and valuable symptom of pulmonary tuberculosis. The mucous membrane is pale.

Hyperæmia may be active or passive. It is readily recognized by the intense redness.

Active hyperæmia occurs with overstrain of the larynx (very frequent and often constant in bass and baritone singers); with irritation from foreign particles, as in "swallowing the wrong way;" inhalation of hot or irritating gases or vapors; in the early stage of inflammations, syphilitic infiltrations, or ulcerations.

Passive hyperæmia occurs in general obstruction to the circulation, as emphysema or valvular lesions; pressure on veins by tumors; forced expiration and holding the breath; in paroxysmal cough, especially whooping-cough. Active hyperæmias lead to catarrhs, passive to œdema.

Acute Laryngitis. Acute laryngitis is an inflammation of the larynx, characterized by a sensation of fulness and dryness in the larynx, with cough, hoarseness, and at times dyspnœa. Several varieties are observed: simple acute laryngitis, laryngitis with great stenosis, laryngitis with membrane, laryngitis with spasm.

It is caused by exposure to cold or by the inhalation of acrid vapors. Excessive use of the voice, particularly in a cold air, may excite an attack. It may be symptomatic of the eruptive fevers, as measles or smallpox, or erysipelas. Its occurrence in the course of chronic diseases must be looked upon with alarm, particularly in cases of Bright's disease, if dropsy is present in other situations.

The attack begins with a feeling of chilliness, followed by *fever* of varying degree, but usually mild. A feeling of pressure and dryness in the larynx, or as if a foreign body were present is complained of. Some *pain* gradually develops in the height of the attack, never so severe as to require an anodyne. From the first there is *cough*. It is dry and hacking, and slightly painful. In the more intense forms the

cough is continuous, disturbing the patient by night and day. Paroxysms occur when the patient speaks or takes food. First the cough is dry; within a short time it becomes moist, and expectoration of a clear, transparent mucus takes place. The mucus may be tinged with blood. At the end of forty-eight hours expectoration grows more yellowish and opaque. The voice may be merely hoarse, or may be lost entirely. Sometimes *aphonia* without general symptoms occurs in acute laryngitis. In *laryngitis sicca* cough and dyspnœa occur in paroxysms and are not relieved until a dry secretion is coughed up. The paroxysms take place at night or in the early morning, and may cause retching and vomiting. It is seen in adults.

Acute Laryngitis with Stenosis. No doubt some of the cases of so-called membranous croup that we see in children are cases of acute laryngitis, with swelling and occlusion of the glottis by congestion and by tough secretion. Œdema may or may not be present. The attack begins with catarrhal symptoms. The child is languid, refuses to eat, has thirst, and some chilliness and rise of temperature. With the slight cough, which may be shrill, there is hoarseness and some difficulty in breathing, but no pain on swallowing. On the second day, or after the lapse of four or five days, during which time mild fever continues, the catarrhal symptoms become more marked. The voice is more hoarse or may be suppressed. The harsh, clanging cough becomes toneless, and soon the sound is suppressed. The respirations are hurried, and dyspnœa is most severe. They are noisy, attended by loud whistling inspiration, snoring expiration. The stenosis is inspiratory, and during the day or in the succeeding twenty-four hours may become very intense. It is attended with violent efforts at breathing and the occurrence of cyanosis in its most aggravated form. The larynx moves up and down, the head is thrown back. There is recession at the root of the neck and along the margins of the ribs and the epigastrium. The lower portion of the sternum may be drawn in. Duskiness of the extremities and of the lips is observed as the stenosis becomes more marked, finally deepening into cyanosis. It may be relieved from time to time by removal of the obstruction, which occurs after cough, vomiting, or change of position. A paroxysm soon recurs. With each paroxysm lividity becomes more and more marked, the respirations continue hurried. The face becomes pale, the extremities cold, and a cold sweat bathes the brow. Restlessness is characteristic. The child tosses about in the bed or from the bed to the arms of the nurse. The heart's action is increased each hour in frequency as the stenosis advances, and becomes weaker. As exhaustion ensues and the symptoms of obstruction become more marked, stupor deepening into unconsciousness develops. Convulsions may occur at the end. The attacks rarely recur if once recovered from. They follow exposure to cold.

If recovery takes place, the child usually becomes more free from dyspnœa, the cyanosis fades, and the restlessness disappears. A prolonged sleep attends the relief, although the voice may remain hoarse or suppressed, and the cough continue many days.

Laryngeal Diphtheria. The same symptoms are seen in cases of membranous croup or laryngeal diphtheria. In the latter

affection there may be a history of exposure or of infection. With the commencement of the attack the patches of diphtheria may be seen in the fauces or nares. If membrane can be secured and a bacteriological examination made, the diagnosis of diphtheria with stenosis is positive; enlarged glands in the neck, with more marked depression, a moderate degree or absence of fever, and occurrence of early albuminuria, also point to diphtheria. The distinction of the two affections is nevertheless quite difficult, and as long as there is a shadow of doubt, for prophylactic reasons the case should be considered one of diphtheria.

Acute Laryngitis, with Spasm. False Croup or Spasmodic Laryngitis. In children, in addition to mild and intense forms of laryngitis, a form is frequently seen associated with spasm of the larynx. The catarrhal symptoms are mild, so that the child seems to be well during the day. Fever is absent and a slight cough or huskiness alone calls attention to the larynx. After the first three or four hours of quiet sleep the child suddenly awakens with a barking cough, sits up and struggles for breath on account of suffocation. The dyspnœa continues for a few minutes to an hour or so, gradually lessening, to disappear entirely as the child lapses into sleep. Throughout the next day the child seems well as on the previous day, and the succeeding night is seized with another attack of "croup." This may occur once or twice during the night. It seems to be influenced by the weather. Damp days and an east wind are provocative of the attack. It recurs frequently during the same season.

Laryngoscopic examination reveals the characteristic appearances seen in cases of acute laryngitis. In children, in whom the disease frequently occurs, such examination cannot well be made.

Inflammation of the Epiglottis. The epiglottis may be inflamed in cases of laryngitis, or become so independently. The sensation of a lump in the throat at the base of the tongue or the top of the larynx is complained of, and pain in attempting to swallow occurs. The pain becomes very intense at times. Fluids cannot be taken, for when the patient attempts to swallow, because the epiglottis does not protect the glottis, the fluid enters. The voice is usually clear throughout the attack, and the general symptoms are not marked.

On laryngoscopic examination the epiglottis is seen as a thick, red tumor. It may be felt by the finger.

Œdema of the Larynx. This condition arises in the course of acute laryngitis, frequently occurs in chronic diseases of the larynx, particularly if ulceration is present, and is a complication of erysipelas and diphtheria. In some cases of Bright's disease it may develop suddenly.

In the course of the above-mentioned disease the onset of the symptoms of laryngeal stenosis may occur suddenly. The voice becomes husky and suppressed, the dyspnœa is very extreme, so that in a few hours grave symptoms of obstruction arise. There is no cough. The patient complains of the sensation of a foreign body and tries to grasp it.

On laryngoscopic examination the epiglottis and aryteno-epiglottidean folds are swollen. The epiglottis can usually be felt by the finger. If so it is of diagnostic importance.

The Diagnosis of Acute Diseases of the Larynx.

Acute affections of the larynx are distinguished from other diseases without much difficulty. To recognize the various forms of acute laryngitis, however, is not so easy. In all there is laryngeal stenosis to a certain degree, and practically the question to answer is, Which form of stenosis is present? The accompanying table shows the differential points for diagnosis. It is seen that the age, occurrence of previous attacks, the character of the general symptoms, the existence of previous laryngeal disease, the association of faucial disease, the presence or absence of membrane, and the results of laryngoscopic examination must be considered before making a positive diagnosis.

Simple Acute Laryngitis.—"Catarrh of Larynx."	*Acute Laryngitis with Stenosis.*
Gradual onset of laryngitis, with dyspnœa very slight or absent.	Gradual onset of laryngitis, but dyspnœa develops to great severity.
All ages.	Children.
Fever of varying degree.	Fever of varying degree.
Dry irritating cough.	Dry cough, often paroxysmal.
May be hoarseness.	Hoarseness.
Pharynx reddened.	Pharynx reddened.
Gradual increase and decline.	Gradual increase, and either death of patient or decline of dyspnœa.
Larynx red and slightly swollen, as seen by laryngoscope.	Same, but swelling much greater.
Acute Laryngitis with Spasm.—Spasmodic Croup.	*Laryngismus Stridulus.—"Child-crowing."*
May be slight hoarseness or cough, or none. Suddenly in night, child wakes with intense dyspnœa and crowing inspiration.	No laryngitis. Sudden attacks of dyspnœa with crowing inspiration, either day or night. Very severe. May be general convulsions.
Children.	Children or hysterical adults.
Temporary high fever.	No fever.
Slight brassy cough during day.	No cough.
May be slight hoarseness in day. Very hoarse in attack.	No hoarseness.
Lasts a few minutes to one hour. May recur or no attack until next night.	Occurs often in rhachitic and hysterical cases.
Slight redness, or nothing seen by laryngoscope.	Ends suddenly, in at most two minutes, and occurs often.
	Nothing seen in larynx.
Œdema of Larynx.	*Membranous Laryngitis.—Croup; Diphtheria.*
Some inflammatory disease of larynx exists.	Epidemic.
Rapid development of dyspnœa, increasing to great severity.	Gradually developing hoarseness and croupy cough, with low fever and lassitude, then development of dyspnœa, gradually and without intermission, as a rule.
All ages.	Children.
Depends on cause.	Low fever and depression.
No cough.	Croupy cough, later suppressed.
No hoarseness.	Very hoarse.
	Fauces red and often with membrane; albuminuria; paralyses.
Increases steadily to climax, then death or decline of dyspnœa.	Increases steadily, broken by intense paroxysms. Either death or gradual improvement.
Epiglottis and aryteno-epiglottic folds swollen, pale, and waxy.	Red, swollen, and membrane.
Foreign Bodies.	*Pertussis.—Whooping-cough.*
During eating or while holding object in mouth sudden dyspnœa, varying in intensity according to object.	Epidemic.
	Bronchitis, with cough developing from one to three weeks. Then dyspnœa caused by severe paroxysms of coughing—absent between them.
All ages.	Children.
No fever.	Only the fever due to bronchitis.
Irritative, expulsive cough.	Intense paroxysms of coughing.
May be hoarseness or not.	No hoarseness.
	Hemorrhages in various places from strain or emphysema.
Cough persists till removal of body, or occasionally the larynx becomes accustomed to its presence, and cough ceases.	May be death from exhaustion, or gradual improvement.
See the foreign body.	Nothing seen, unless slight laryngitis.

Chronic Laryngitis.

Chronic laryngitis either originates in an acute attack or comes on slowly. Prolonged use of the voice in a higher key than natural or in the open air, the use of alcohol, constant exposure, are exciting causes. It is symptomatic of syphilis and tuberculosis. It frequently results from inflammation of the upper air-passages, particularly chronic pharyngitis. It occurs after middle life more frequently, and usually in the male sex. There is discomfort on long speaking, with dryness and tickling. At first the secretion of mucus is very slight, but after hawking and coughing, it increases in amount. Hoarseness occurs, and if the patient is careless or persistent in the baneful occupation, complete aphonia may arise. The voice is clearest in the morning, after expectoration of the mucus that accumulated in the night, but becomes husky toward night. The aphonia may occur in paroxysms, relieved by coughing up of dry secretion. The cough is never severe. The sputum is small in amount, glairy, and often in little balls or crusts.

Laryngoscopic Examination. Hyperæmia and swelling of varying degree of the epiglottis, the outer arytenoid space, and the false cords are seen. The cords may be uneven, or granular from nodes. Fine threads of secretion, or little balls of mucus, may also collect. Fissures or erosions are seen on the cords and in the folds. In the dry form of chronic laryngitis, the mucous membrane is pale and thin, and crusts form.

Acute Submucous Laryngitis.

The inflammation extends to the submucous cellular tissue. It arises in the course of acute laryngitis, and is the form seen in traumatism, or from burns and scalds. The symptoms are those of intense laryngitis with stridor. They increase in severity until stenosis arises. If the under surface of the cords is affected, death will occur from asphyxia. Sometimes the inflammation is circumscribed and followed by development of an abscess.

On laryngeal inspection the *diffuse* form cannot be distinguished from ordinary laryngitis. The *circumscribed* form is recognized by a swelling on the top of which the yellow point, due to the suppuration, gradually appears. In the *hypoglottic* form, or so-called *œdema* of the *glottis*, a round, fixed swelling is seen on each side below the vocal cord, almost entirely occluding the larynx.

The *chronic form* of *submucous inflammation* of the larynx is usually seen in drunkards, and is recognized usually by the laryngoscopic examination. The symptoms are those of slight stenosis. On inspection a dirty-red diffused or circumscribed swelling of some part of the larynx is observed. It may be seen on the epiglottis, or the arytenoepiglottic folds below the cords.

Phlegmonous Laryngitis or Perichondritis.

Inflammation about the cartilages is usually phlegmonous in character, and leads to the formation of abscess. The collateral œdema is so

great as to cause some obstruction, with cough and hoarseness. On palpation, the larynx is extremely tender. The pain is increased by movement of the larynx, and occurs in speaking or swallowing. If the inflammation involves the arytenoid cartilages, pain extends toward the ear, the vestibule is swollen, the cartilage fixed. On the other hand, when the cricoid is diseased, there is pain on swallowing of solid food on account of interference with the muscular attachments, dyspnœa, and paralysis of the posterior crico-arytenoid muscles. Inflammation of the thyroid cartilage may open externally or internally. In the latter case the abscess can be seen in the larynx.

An examination by the laryngoscope shows swelling or œdema so great that the parts cannot be well outlined. Discharge of pus and necrosed cartilage confirms the diagnosis. By means of a sound the cartilage can be detected, giving further proof of the presence of the disease.

Neuroses of the Larynx.—Laryngismus Stridulus.

Laryngismus Stridulus, or spasm of the glottis, is seen usually in children that are poorly nourished. It is of frequent occurrence in *rickets*, indeed its occurrence points very strongly to the possibility of that disease being present in children in whom otherwise the manifestations are obscure.

The symptoms occur suddenly, and are very alarming. The child awakens in the night, and after a few short whistling inspirations, sudden cessation of breathing takes place. The child is seized with terror, which is depicted on the countenance; the eyes stare, the face is pallid at first, but rapidly becomes livid. The alæ nasi are extended, the head is thrown back, and the spine arched. A cold perspiration breaks out over the forehead. Carpo-pedal spasms may occur and the urine and fæces be discharged involuntarily. After a period varying from a few seconds to at the furthest two minutes, the child draws two or more deep, noisy inspirations, each one lessening in depth and sound, when color returns to the face, the cyanosis gradually disappears, and the child becomes tranquil.

In mild forms the child "catches its breath." It holds its breath, and then makes a noisy inspiration.

The attacks of laryngismus stridulus are more rare in adults. They may occur in hysterical subjects. In the attack there occurs a series of long, harsh, whistling or stridulous inspirations, followed by short, noisy expirations. Rarely is there complete closure of the glottis.

In both children and adults general convulsions may occur during the attack, or carpo-pedal spasms alone may be seen. In adults the convulsions occur only in hysterical subjects.

Spasm of the glottis is a frequent complication of diseases of the larynx. It is due to peripheral irritation in the idiopathic form.

The diagnosis of laryngismus stridulus is based upon the absence of laryngeal symptoms prior to the attack, the absence of cough or hoarseness, and the complete disappearance of all laryngeal symptoms when the attack subsides. The absence of pain and fever and of laryngo-

scopic signs is noteworthy. This applies, of course, to spasm that occurs independently of laryngeal disease.

Paralyses of the Laryngeal Muscles.

They are divided for convenience into groups. The *symptom* is dysphonia, which, with laryngoscopic appearances, leads to the recognition of the paralysis.

1. Paralysis of the Tensors of the Cords. The crico-thyroid muscle is paralyzed; the superior laryngeal nerve, which supplies the muscle is concerned. The *voice* is deep and rough, and incapable of producing high tones. Usually, the whole nerve is involved, and the result is *anæsthesia* of the larynx and *paralysis* of the *epiglottis* also.

Laryngeal Examination. The *epiglottis* is fixed and back against the the tongue. The *glottis opening* is a wavy line.

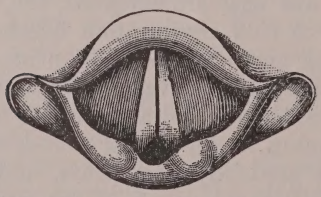
Causal disease. The condition described occurs almost exclusively after diphtheria.

2. Paralysis of the Closers of the Glottis, or Adductors of the Cords. The muscles involved are the crico-arytenoideus lateralis, arytenoideus transversus, and the thyro-arytenoidei internus and externus. The nerve is the recurrent laryngeal.

The symptoms are *complete aphonia*, coming suddenly, and often as suddenly going.

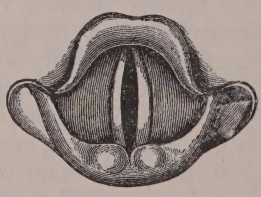
Laryngeal Examination. During phonation the cords remain in the inspiratory position. The paralysis may affect one or both sides.

Fig. 29.



Paralysis of the arytenoideus transversus in phonation. (Gottstein.)

Fig. 30.



Paralysis of the thyro-arytenoideus internus in phonation. (Gottstein.)

Sometimes the arytenoideus transversus alone may be affected. Then there is hoarseness or aphonia. The anterior portions of the cords come together in phonation, but the posterior portions do not, leaving a triangular opening posteriorly. (See Fig. 29.)

Or, the thyro-arytenoideus internus may alone be affected. There is then dysphonia or aphonia, as before, but the cords come together at both extremities and remain apart in the middle, forming an oval opening. (See Fig. 30.)

Causal disease. These paralyses occur in hysteria, catarrh, or severe overstrain of the voice.

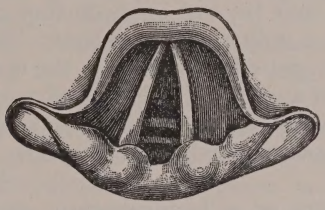
3. Paralysis of the Openers of the Glottis, or Abductors of the Cords. The muscle affected is the crico-arytenoideus posticus, and the nerve is the recurrent laryngeal.

Symptoms. When one side is affected the respiration is free, but there is stridor on forced inspiration. The voice is harsh.

Laryngeal Examination. One cord remains in the middle line. (See Fig. 31.)

When both sides are affected there is gradually developing inspiratory dyspnœa with stridor. The voice is nearly normal.

FIG. 31.



Paralysis of the left recurrent nerve: inspiration. (GOTTSTEIN.)

Laryngeal Examination. The glottis is a narrow cleft which becomes still narrower on inspiration.

COMPLETE PARALYSIS OF THE RECURRENT LARYNGEAL NERVE.

Symptoms. Unilateral paralysis. A weak toneless voice which goes into a falsetto when the patient endeavors to speak loud.

Laryngeal Examination. The cord and arytenoid body are in the cadaveric position, viz., half-way between the phonating and the inspiratory positions. In phonation the other cord passes beyond the middle line, and the glottis is slanting. The edge of the paralyzed cord is excavated.

Bilateral paralysis. Aphonia and inability to cough and expectorate.

Laryngeal Examination. Both cords are in the cadaveric position and their edges excavated.

The adductors are usually paralyzed before the abductors, and one can see all the intermediate stages by close watching.

Causal disease. The conditions which give rise to the paralysis are numerous. It may arise from simple catarrh or from hysteria. More often it is due to pressure on the vagus or recurrent laryngeal, or some disease affecting these nerves or their roots.

The causes of pressure are: Aneurism of the subclavian or aorta, mediastinal tumor, tubercular bronchial glands, a tubercular apex of a lung, cancer of the œsophagus, goitre, or carcinoma of the pleura.

The diseases are: Diphtheria, tumor, softening or hemorrhage into the brain, bulbar paralysis, neuritis, typhus, cholera, variola, articular rheumatism, toxæmia (?), sclerosis of the cord, progressive muscular atrophy, and paralytic dementia.

Tumors of the Larynx.

Both benign and malignant growths are seen. They give rise to the same group of *symptoms.* At first *dysphonia* or *aphonia* takes place. The impairment of voice may continue for a long period of time before

dyspnœa arises. This develops very gradually, and in some few cases is attended by an irritative *cough*.

The general symptoms are not marked in benign cases. In the malignant forms they are pronounced, but characterized by the development of cachexia later than in carcinoma elsewhere.

The most common form of the benign growths is *papilloma*. The growth may spring from the true or false cords, the aryteno-epiglottic ligaments, rarely the posterior surface of the epiglottis. The tumor has a broad base. There may be one only, or they may be multiple, and may vary in size from a split pea to a walnut. Three varieties are met with: 1. Small warty growths, usually on the cords, dark red in color and seldom larger than a bean. 2. Groups of raised white papillæ on a broad base, also growing on the cords. 3. Large, red, mulberry or cauliflower-shaped growths, partly villous, partly warty, which fill up the whole larynx.

Fibroma. It appears as a hemispherical, pedunculated tumor of dirty-white, reddish, or dark-red color, more or less dense in consistency. It is usually single, and grows most frequently from the cords. When seen in its smallest size, it is known as the "singer's node." It may be as large as a hazelnut.

Malignant Tumors. In addition to the symptoms indicated in benign tumor, pain and hemorrhage occur.

Both *carcinoma* and *sarcoma* are found; the latter is very rare.

Carcinoma. The most common form is the epithelioma, although the medullary and scirrhus have been described. The epithelioma is seen as a circumscribed, hemispherical, warty or cauliflower-like formation, varying in size, or as a knotty infiltration projecting into the larynx. The medullary form is larger, soft and bloody, and rapidly ulcerates. Scirrhus is firm and hard. The structure of the larynx is gradually invaded, with necrosis of the tissues. Perichondritis and abscess frequently ensue.

In carcinoma of the *cords* two modes of growth are seen.

In the polypoid form the tumor develops on the cord like a warty growth, sometimes papillary and of a reddish gray color. In diffused cancer of the cord the structures are red and knotty and the tissues invade the surrounding tissue without distinct demarcation.

Sarcoma. The tumor has a broad base, is shining in appearance, and sometimes lobulated. Sometimes the structure is dark red or yellow.

The *diagnosis* of malignant disease of the larynx is based upon the association of symptoms of laryngeal disease with pain, and with the characteristic appearances found on inspection, occurring after the middle period of life, lasting from six to nine months only, with the development of cachexia and emaciation without fever. Enlargement of the cervical glands points to cancer. Simple and syphilitic perichondritis must be excluded.

Tuberculosis of the Larynx.

The existence of primary laryngeal tuberculosis is doubtful. It cannot be proven clinically, and the majority of cases, at least, are sec-

ondary to tuberculosis of the lungs. The manifestations of tuberculosis of the larynx may be either a simple persistent catarrh, an infiltration or ulceration. The symptoms vary according to the lesion.

a. CATARRH. There is a slight hoarseness and the voice tires easily. Often paræsthesia or peculiar sensations in the larynx are present. Cough, when due to this alone and not to the process in the lungs, is short and dry.

Laryngoscopic examination is either negative or shows a peculiar anæmia of the mucous membrane.

b. INFILTRATION. At first the symptoms are those of simple catarrh, then the alteration of the voice increases even to aphonia; there is a feeling of dryness or soreness in the larynx, and dysphagia. The cough is very slight and is usually wholly disguised by the cough due to the disease in the lungs. There is some difficulty in expectoration.

Laryngoscopic Examination. Attention is first attracted by the marked anæmia of the mucous membrane. At first there are slight intumescences of tubercular infiltration, not well outlined, and gray in color. They are most frequently found in the inter-arytenoid space, less often on the false cords and arytenoid cartilages, rarely on the epiglottis.

1. A hill-like prominence between the arytenoid cartilages either in the middle or on one side. In phonation it presses between the cords.

2. When a false cord is affected the whole of it is usually infiltrated, forming a tumor-like swelling which often hides the vocal cords.

3. Vocal cords. Usually only one cord is at first affected. It is thickened and the free border is red. Sometimes the free edge seems split. The infiltration may extend to the subcordal region and cause a hypoglottic laryngitis.

4. Epiglottis. Infiltration of the epiglottis is rarer than œdema after ulceration, and care must be taken not to confound these conditions. The whole epiglottis, or only portions of it, may be affected. It is thickened and curled upon itself, and not freely movable.

5. Arytenoid cartilages. They appear enlarged and puffy, and often fixed from perichrondritis.

c. ULCERATION. The symptoms are the same as those of infiltration, but the dysphagia and pain are greater. It occurs in the

1. Inter-arytenoid space. The mucous membranes are notched with irregular projections. When the ulcer is visible it is irregular and of a dirty-gray color.

2. False cords. The ulcers are flat and aphthous with a pale-white base and a membranous deposit. The mucous membrane sometimes appears sieve-like.

3. Aryteno-epiglottic ligaments. The ulcers are superficial and run lengthwise of the ligament.

4. Vocal cords. The ulcers are either on the upper surface or on the edge of the cords. The former are superficial and seldom destructive. Those on the edge are either small separate ulcers or long ones, affecting the whole border. The circumscribed ulcers occur usually at the posterior portion of the cord and on the processus vocalis. The ulcers of the whole border are often very destructive.

5. Epiglottis. Tubercular ulcers of the epiglottis occur only on its laryngeal side. They are either aphthous and superficial, or deep, and arise from the breaking down of previous infiltration. Sometimes tubercles can be seen at the edge of the ulcers, but they are of no diagnostic value, as similar nodes are seen with non-tubercular ulcers. The epiglottis is usually thickened and œdematous.

Diagnosis. Tuberculous ulcer occurs most frequently in the male sex, and during the period ranging from eighteen to thirty years of age. If the symptoms develop in the course of phthisis, or in case that affection cannot be recognized, if there is a history of infection, or exposure, and if bacilli are found in the sputum, the diagnosis is not difficult. A portion of the diseased mass may be removed for microscopic examination or inoculation. In examining the secretion for tubercle bacilli, it is to be remembered that the exudation may have been brought up from the lungs. The examination in cases of phthisis is of little practical value, except to determine whether the ulceration present may be syphilitic and grafted upon a tuberculous disease of the lungs. Enlargement of the glands of the neck is often present, but not diagnostic.

Fever is present, and, indeed, may be an important diagnostic feature in cases of doubt. The temperature should be taken every two hours, for the morning or evening exacerbations may not be present. Emaciation ensues, and sooner or later the hectic phenomena and signs of tubercle in other structures arise. When tuberculous ulceration of the larynx occurs in the course of local pulmonary tuberculosis the disease runs a much more rapid course.

The laryngeal symptoms are not diagnostic. Pain may be the most distinct. The appearances observed by the laryngoscope are more characteristic. Local anæmia with paræsthesia, paresis of the cords, and short cough, or an obstinate diffuse catarrh, are suspicious symptoms. The peculiar ridged infiltration between the arytenoids is almost invariably tubercular.

Isolated thickenings anywhere in the larynx which shade gradually off into the normal tissue can be only tuberculous or syphilitic. The regularity and number, with anæmia and lack of inflammatory signs, will usually distinguish the tuberculous from the syphilitic. The ulcers are non-erosive. Syphilitic ulcers do not often occur, except on the edge and lingual side of the epiglottis and on the cords. They extend more rapidly than the tuberculous, and may be continuous with ulceration in the pharynx. The area of ulceration may extend to the base of the tongue, which is very infrequent in tuberculous disease. In syphilitic ulceration scars or cicatrices are seen; they are absent in the tuberculous form. Laryngoscopic examination in tuberculous ulceration is difficult, causing great pain; in syphilis comparatively little pain attends examination.

Syphilitic Affections of the Larynx.

Mucous patches, papules, infiltrations, or gummata may be present in the larynx for some time with no symptoms whatever. Usually a change in the voice is the first symptom noticed, due either to the catarrh or to

ulcers, scars, infiltrations, or gummata affecting the cords. There is often a feeling of pressure or a tickling sensation. Pain is not usual, and when present is very slight. Dysphagia occurs only when the epiglottis is extensively ulcerated. There is little or no cough.

Laryngoscopic Examination. The appearances vary with the condition.

1. *Catarrh.* Nothing characteristic to be seen.

2. *Mucous patches.* These are flat elevations of 3 to 7 mm. diameter, oval or circular, and of a whitish-gray color. When the epithelium is lost they appear yellow and purulent. There is no tendency to ulceration, and the patches soon disappear, even without treatment. They occur usually from three to nine months after the infection.

3. *Infiltrations.* Usually these are overlooked, as they produce no symptoms. They are diffuse thickenings in various parts of the larynx, most often on the epiglottis. This may be uniformly thickened or only a part of the edge. The cords may be so swollen as to cause dyspnœa. Usually an ulcerated spot is seen in the centre of the infiltration. The mucous membrane is either normal or reddened. Infiltrations appear three to four or more years after infection.

4. *Gummata.* They appear as round prominences of the same color as the surrounding tissue. They occur on either side of the epiglottis, on the ary-epiglottic folds, often in the inter-arytenoid space, on the false cords, and on the under surface of the vocal cords. If they break down deep ulcers form, leading to extensive destruction of the parts.

5. *Ulceration.* Syphilitic ulcers are circular, deep, with a sharp border and inflammatory areola, and overlaid with a whitish-yellow deposit. They develop from an infiltration or a gumma, and not on an unchanged surface. Ulcers on the upper surface of the epiglottis are always syphilitic.

The *diagnosis* rests upon the history of infection, the objective signs of syphilis indicated by pigmentation or recent eruption, scars, periostitis or nodes on the bone, and enlarged glands. The laryngeal symptoms are not diagnostic, save that pain is absent in spite of extensive ulceration, while difficulty of deglutition on account of food entering the larynx is of frequent occurrence. The larnygoscopic appearances, as indicated above, are characteristic of this affection. In obscure cases the distinctions spoken of in tuberculosis are of diagnostic value.

Although the patient may be broken down and cachectic the febrile range is not high, unless perichondritis occurs, or the onset of pneumonia arises on account of food in the air-passages.

Lupus.

In this affection, probably tuberculous, there is soreness and slight dysphagia with slight hoarseness, deepening to dysphonia or even aphonia. In the later stages dyspnœa can arise from infiltration or *scar contractions.* Lupus is usually present also in the *skin* of the *face* and in the mouth and pharynx.

Laryngoscopic Examination. Isolated or grouped *nodes* flowing

together into patches are seen most frequently on the epiglottis. Later *ulceration* occurs with loss of substance and *scar formation.*

Lepra.

The symptoms are dysphonia and dyspnœa. Usually lepra is present elsewhere.

Laryngoscopic Examination. The epiglottis is swollen, red, and vascular, the arytenoid bodies and false cords dark red to bluish, the cords injected and thickened. Nodes from the size of a pin-head to that of a pea are seen on epiglottis, arytenoid bodies, and false cords. Then follow ulceration and loss of substance.

Foreign Bodies.

These may be particles of food, false teeth, pins, or almost anything small enough to enter the larynx, which could by any possibility be placed in the mouth. The symptoms are cough, often with spasm of the larynx and dyspnœa. There is pain only when the foreign body is sharp and capable of injuring the mucous membrane. Hoarseness is observed when the cords are interfered with.

Laryngoscopic examination is not always possible on account of the reflex spasm. When examination is possible the body can usually be seen.

The Larynx in Other Diseases.

In Nervous Diseases. Laryngeal symptoms due to lesions of the nervous system. (See Cerebral Localization.)

Cerebral hemorrhage. 1. Aphasia. The movement of the muscles is normal, but they cannot be controlled by the will. Caused by hemorrhage in the cortex or along the course of connective fibres.

2. Recurrent paralysis. Due to hemorrhage in the medulla.

3. Symptoms of bulbar paralysis. Same cause.

Encephalomalacia. (Softening.) When in the brain, aphasias result; when in the medulla, bulbar symptoms.

Tumors of Cerebrum. The symptoms are, according to location, aphonia, aphasia, or paralysis of the cords.

Bulbar Paralysis. We have, of course, the other symptoms of the disease. The voice becomes weak and monotonous without modulation. High tones cannot be made. It progresses to hoarseness and finally aphonia. Particles of food and drink enter the larynx. Paresis or paralysis of the cords.

Multiple Sclerosis. The speech is slow, uncertain and scanning, later hoarse. Laughing and crying are accompanied by peculiar yawning inspirations.

Laryngoscopic Examination. Slight paresis of the cords is seen.

Posterior Sclerosis (Tabes). The muscles act very slowly. Sometimes symptoms of irritation, as tickling or burning in the larynx, with a dry cough, occasionally severe paroxysms of coughing even to spasm of the larynx, occur. In rare cases a phonetic spasm has been observed. Less often pareses or paralyses of the various muscles occur, most fre-

quently the posticus, next the recurrent. Sensibility may or may not be disturbed.

Amyotrophic Lateral Sclerosis. There is a mixture of bulbar with spinal symptoms. (See Sclerosis.)

Progressive Muscular Atrophy. Very late occurs this same mixture of symptoms.

Paralytic Dementia. There may be disturbances in articulation with paresis and paralysis of the cords.

Chorea. There may be a tremor of the cords from under-tension, but probably no true choreic movements.

CHAPTER II.

DISEASES OF THE LUNGS AND PLEURÆ.

THE various affections of the lungs occur without any change in the volume of air in the lungs, or are attended by an increase or diminution in the amount of air.

I. Diseases with Normal Amount of Air.

AFFECTIONS OF THE BRONCHIAL TUBES, EXCEPT ASTHMA.

II. Diseases with Increased Amount of Air.

ENLARGEMENT OF THE CHEST. The enlargement with increased amount of air may be unilateral or bilateral. It seems anomalous that the more air in the thorax, the greater need for air and hence the occurrence of dyspnœa.

1. Asthma.
2. Emphysema.

III. Diseases with Diminished Amount of Air.

A. THE CONSOLIDATIONS. The consolidations may be local, unilateral, or bilateral.

1. The congestions.
2. Pulmonary embolism and thrombosis.
3. Pneumonia.
4. Broncho-pneumonia.
5. Chronic interstitial pneumonia.
6. Pulmonary tuberculosis.
7. Abscess of the lung.
8. Gangrene of the lung.
9. Collapse of the lung.
10. Cancer and other new growths of the lung.
11. Hydatid disease of the lung.

B. DISEASES OF THE PLEURA.

1. Diminished amount of air from inhibition of movement on account of pain.
2. Diminished amount of air from the physical condition within the thorax.

The lungs are composed of a relatively small amount of tissue. They are made up of tubes and canals. The tissue which composes the structure of the lungs independent of the canals, the connective tissue, is liable to the same morbid processes that affect it in other situations. But, curiously, it is not often subjected to irritants on account of which acute inflammation takes place, while chronic inflammations occur secondarily, in the large majority of cases, to processes in the channels.

Diseases of the lungs therefore are the diseases of its channels, and the symptoms that arise are due to morbid alterations of them (1) by processes common to the structure of such channels, and (2) by obstruction of them. The channels are three: first, for the passage of air; second, for the flow of blood; and third, for the flow of lymph.

Symptoms due to the Morbid Process. The air-tubes are lined with mucous membrane which is subject to morbid processes that attend any such lining—congestion, or acute and chronic inflammation—with a flux as the characteristic symptom. The muscle and elastic tissue of the canal become involved in the process. The former undergoes spasm with or without mucous membrane inflammation (asthma). Grave disaster does not arise until degeneration takes place—then the power of confining the air or driving it out is lost, and emphysema results.

In the blood canals, hyperæmia (congestion), embolism and thrombosis, and secondary œdema take place; while in the lymph canals, inflammation (acute and chronic pleurisy), and transudation (hydro- or hæmothorax) take place. Now, the symptoms that arise in each or all of the above processes—pain, local discomfort, mucous or purulent discharge, serous or purulent exudation, and fever—are not different from those which are found in similar tissues in other localities.

Symptoms due to Obstruction. But in addition to the group of symptoms thus indicated there is a group due to obstruction of the various channels, and hence, interference with the function of the lungs. The symptoms are purely mechanical.

1. *Dyspnœa* occurs from obstruction of either canal. It is as pronounced in asthma or capillary bronchitis as in embolic obstruction (fat embolism), or congestion and stasis in the bloodvessels. It occurs when the canals are occluded by extrinsic causes—foreign bodies in the bronchi, or pleural effusions.

2. *Cyanosis.* As a sequence of the above symptom we have another vivid picture—the development of cyanosis from interference with aëration.

Symptoms from Other Causes. Other structures (the bony thorax and its muscles) are required for the performance of the function of the lung, the aëration of the blood.

Of these more particularly we have: first, muscles, to hasten the movement of the air; and second, a nervous mechanism to control the muscles. Inactivity of the former, from pain, from debility, or from paralysis through disease of the nerves, practically occludes the canals, for the normal contents cease their movement or lessen its speed, and therefore the amount of air is lessened—hence, again, dyspnœa. The nervous mechanism not only controls the large muscles of the exterior through a centre stimulated or depressed by various influences, chiefly the blood, but also receives and sends impressions to the muscles of the canal, on account of which we have (*a*) *cough* or (*b*) *bronchial spasm* with dyspnœa. This nervous mechanism, with its centre of control, is in relationship with higher and lower centres, and the nerve that connects it with its organ supplies other organs or anastomoses with other nerves. Hence, we may have: *A.* A *central affection,* causing

pulmonic symptoms from these causes—1. Because higher centres influence the lower pulmonary centre, as we see in hysterical cough, or emotional cough, and in asthma. 2. Disease affects the region of the centre, as in tumor or in bulbar or glosso-labio-laryngeal paralysis. 3. Irritants act upon the centre, as urea, exciting uræmic asthma. *B.* An affection of the *nerve trunk*, as from the pressure of an aneurism or morbid growth. *C. Reflex influences* through the pneumogastric and correlated nerves. The asthma of nasal disease or of peripheral irritation elsewhere, and reflex cough, is of this nature. *Corollary:* Lung symptoms, chiefly dyspnœa and cough, may be due to local causes (affections of the muscles), or to causes at a distance, operating directly through the pneumogastric centre, or the nerve trunk, or by anastomoses in a reflex manner. The practical deduction is, to look further than the lungs in the investigation of pulmonic symptoms. Lung symptoms are not so often expressions of disease in other parts, nor do diseases of that organ so often have their expression in other organs, as is true in gastric diseases.

Affections of the Pleura. In diseases of the *pleura*, one side is usually affected, but whether the disease is unilateral or bilateral we have simple inflammation, and inflammation with exudation into the pleural cavity. In both forms there is diminution of movement, and hence less air entering the affected lung, although the cause for the diminution in the amount of air is different in each case. In *acute inflammation*, the lessened amount of air is present because of physiological reasons. The movement of the affected side is inhibited by pain, hence diminution of expansion and lessened ingress and egress of air follow. It is true, enfeeblement of breath-sounds and fremitus, with diminished expansion, alone indicate the diminution. On the other hand, in acute inflammation with exudation, the diminution in the amount of air occurs on account of physical reasons. The effusion encroaches upon and causes diminution of the air-space, and hence lessens the amount of air. It will be remembered that the physical signs of diminution in the amount of air from effusion are quite distinct from the physical signs due to consolidation.

The *Lungs* and *Heart*. The relationship of the pulmonary vascular channels to the remainder of the circulation is very close. Overfilling of the pulmonic bloodvessels, and hence dyspnœa, may be due to alterations or changes in the central pump, the heart; or in the vessels between—as from the pressure of an aneurism. The nature and importance of any lung symptoms cannot be appreciated without an investigation of the heart and the blood-ways. Many pulmonic congestions are due to dilatation of the heart, and are relieved by digitalis. At the other end of the beam, it may be noted that lung diseases cause heart disease; from backward pressure of blood columns in overdistended vessels, a dilated right heart follows.

Space forbids tracing out the effects of the blocking of channels, but it is suggestive that all the aëration of the body takes place through the first set of tubes, that all the blood of the body passes through the second, and that the third is an enormous drainage area of lymph. The student can readily appreciate how profoundly diseases of the lungs

must affect the general system. Apart from the nerves, the tie that binds the other organs to them is the blood. As the lungs enrich it with oxygen, so the organs act with vigor. Imperfect oxygenation soon causes diminution of all function, with the secondary effect on the blood of the production of anæmia.

Infectious Diseases. The lungs are subjected, in a high degree, to one group of processes—those of infection. Pronounced symptoms due to the process and to the blocking of channels are produced. They are seen in tuberculosis, pneumonia, the bronchitis of infectious diseases, the pleurisy of septic processes. The general symptoms belonging to such processes are detailed elsewhere.

Relative Value of Subjective and Objective Symptoms. The subjective symptoms are few, and, as will be seen later, are common to so many diseases that they are of little diagnostic value. Fortunately the physics of the lungs come to our relief. Disturbance of this respiratory function causes a physical change. The effect of the occlusion of channels is mechanical or physical, and also causes a physical change in the lung. 1. The objective symptoms are possible because of the physiological movement of air. Sounds attend the movement of air in health; no sounds occur if the air movement is checked, or abnormal breathing and new sounds (râles) are created. 2. They are possible because of physical changes in the structure. Air is replaced by solid structure; the physical condition of the lung changes. The objective signs of these conditions are determined by means required to secure physical data: inspection, palpation, percussion and auscultation.

Diagnosis. The diagnosis of disease of the lungs is attained by the collection and consideration of data obtained by inquiry and data obtained by observation. By inquiring we learn, first, the history of the case; second, the subjective phenomena. By observation the objective phenomena of the disease are determined. The objective phenomena are secured, first, by physical examination; second, by an examination of the sputum, and third, by an examination of the fluids secured by puncture. The examination of the sputum and of aspirated fluids is made with the microscope and by bacteriological methods. For convenience the objective phenomena will be considered first.

It is not generally difficult to distinguish diseases of the lung from affections of other structures. It is true pleurisy and pleurodynia are often distinguished with difficulty. We are called upon, also, to decide between pleurisy and sub-diaphragmatic inflammation, a pleural and hepatic inflammation, a pleuritis and pericardial inflammation, and between cardiac and pulmonary disease, especially when both are present and it is desirable to determine the primary affection. The contiguous relations of the organs make this necessary, but with care in ascertaining the history and the subjective and objective symptoms the distinction may not be difficult.

In chronic disease, affections of the lungs, of the mediastinum, and of the great vessels must be distinguished from one another. An aneurism may simulate chronic phthisis or mediastinal disease.

The Data Obtained by Observation.

THE OBJECTIVE SYMPTOMS. By physical examination of the lungs we ascertain—1, their degree of activity (movement); 2, the physisical condition of the parts subjected to examination: the disease is not diagnosticated. If abnormal signs are detected they simply indicate an abnormal physical condition of the part. As the lungs in health contain air, any physical change that takes place causes either an increase or diminution in the amount of air. This may be general (bilateral), or limited to one side (unilateral), or to a smaller area (local). In an examination of the lungs we might be content to answer the question, Is there an increased amount of air, or a diminished amount in the parts suspected to be the seat of disease? A correct answer to this question, and to an inquiry as to the cause of the increase or diminution, would explain any abnormal physical condition. The answer can be determined by percussion, a method employed to detect such physical condition under any circumstance. But fortunately, as adjuncts we have the phenomena that can be elicited by means of inspection, palpation, and auscultation. The latter methods elicit control data on account of the movement of the lung, and because sound is created by the movement.

Value of Inspection and Palpation. Too much emphasis has been placed in the past on auscultation and percussion in the study of the diseases of the lung. It has grown to be too much the habit to rely on these methods to the exclusion of the simpler and yet at the same time fully as valuable methods—inspection and palpation. The latter have been employed for a long time in the study of the objective phenomena of disease. The former are comparatively modern methods. They required special cultivation of the senses not usually employed in observation, and exhaustive comparative research, to put the findings on an accurate basis. The impetus derived from this study has caused undue stress to be placed upon them as methods of diagnosis. The pernicious habit of examining the patient without removal of clothing, on account of haste upon the part of the physician, or improperly applied modesty upon the part of the patient, has also led unfortunately to the neglect of inspection and palpation. It is proper to insist that the data derived by inspection and palpation are as important and valuable as those derived by other means. The facts derived through them are even more suggestive or diagnostic of physical conditions. The phenomena observed are more positive and surrounded by fewer qualifications.

The Regions of the Chest. For the purpose of bearing in mind the relations of the organs to the surface of the chest, and the localization and proper recording of the seat of the disease, the chest is divided into regions. The regions correspond to anatomical points on the surface of the chest, and are subdivided by transverse and vertical lines. Knowledge of the landmarks which on the surface indicate the position of the parts underneath is of great importance in diagnosis. The *regions* in the anterior portions of the chest are: The supra-

clavicular region, above the clavicle; the infra-clavicular region, below the clavicle, extending to the third rib; the mammary region, from the third to the sixth rib. In the axilla two regions suffice—the upper and lower—the position of the disease being more definitely determined by association with ribs and interspaces. Posteriorly the regions are: the supra-scapular, above the scapula; the scapular region, and the infra-scapular region; the region between the scapula and the spine is known as the interscapular region. The *vertical lines* are to the right and left of the median line: (1) the parasternal line, which is drawn midway between the edge of the sternum and the second line, which is (2) the mid-clavicular line, drawn from the middle of the clavicle, generally passing through the nipple in males; (3) the anterior axillary line, drawn from the anterior fold of the axilla; (4) the mid-axillary line, from the centre of the axilla; (5) the posterior axillary line, from the posterior fold of the axilla. In the back one line is sufficient—the scapular line, drawn through the angle of the scapula when the arm is at rest at the side of the patient. For transverse lines the ribs and interspaces are used. In this way the exact location of a diseased area can be indicated. In order that accuracy may attend its localization, knowledge of the methods of determining the landmarks, and especially counting the ribs, is essential.

The Angles of the Thorax. The *costal* angle is the angle of the rib. It varies during the act of respiration. In inspiration the rib rises as the sternum projects, and apparently elongates; the angle becomes more obtuse; in expiration the sternum falls, the ribs become more slanting, and the angle is more acute.

The *epigastric* angle. This angle is formed by the convergence of the ribs of both sides to the xiphoid cartilage of the sternum. On inspiration, it is obtuse, increasing as the ribs rise; in expiration it is more acute.

Method of Counting Ribs and Interspaces. The first rib corresponds to the clavicle; the first interspace is the region between the clavicle, or first rib, and the second rib; the subsequent number of an interspace corresponds to the number of the rib above it. The following from Holden is of great importance to remember, particularly when the ribs of fat persons are counted:

a. The finger passed down from the top of the sternum soon comes to a transverse projection, slight, but always to be felt, at the junction of the first with the second bone of the sternum. This corresponds with the middle of the cartilage of the second rib.

b. The nipple of the male is placed in the great majority of cases between the fourth and fifth ribs, about three-quarters of an inch external to their cartilages.

c. The lower external border of the pectoralis major corresponds with the direction of the fifth rib.

d. A line drawn horizontally from the nipple round the chest cuts the sixth intercostal space midway between the sternum and the spine. This is a useful rule in tapping the chest.

e. When the arm is raised, the highest visible digitation of the serratus magnus corresponds respectively with the seventh and eighth ribs.

f. The scapula lies on the ribs from the second to the seventh, inclusive.

g. The eleventh and twelfth ribs can be felt, even in corpulent persons, outside the erector spinæ, sloping downward.

h. One should remember the fact that the sternal end of each rib is on a lower level than its corresponding vertebra. For instance, a line drawn horizontally backward from the middle of the third costal cartilage, at its junction with the sternum, to the spine, would touch the body, not of the third dorsal vertebra, but of the sixth. Again, the end of the sternum would be at about the level of the tenth dorsal vertebra. Much latitude must be allowed here for variations in the length of the sternum, especially in women.

It is important to recognize the relation of the ribs to the vertebræ. The first rib articulates with the first dorsal vertebra, which can be located by the position of the prominent spine of the seventh cervical vertebra; even in very fat people this prominence can be recognized. The remaining ribs, except the eleventh and twelfth, have facets of articulation on two vertebræ: as the second rib, with the second and third vertebræ. The eleventh and twelfth articulate with the last dorsal.

Topographical Anatomy. The following anatomical points are worthy of remembrance:

The top of the sternum is on a plane with the lower border of the second dorsal vertebra behind. The junction of the first and second portions of the sternum is known as the angle of Ludwig. It is opposite the middle of the second rib, and is on a plane with the lower border of the fourth dorsal vertebra. The junction of the body of the sternum to the xiphoid cartilage is on a plane with the lower border of the eighth dorsal vertebra.

The apex of the diaphragm is on a level with the eighth dorsal vertebra.

The trachea bifurcates at the plane which includes the angle of Ludwig and the fourth dorsal vertebra.

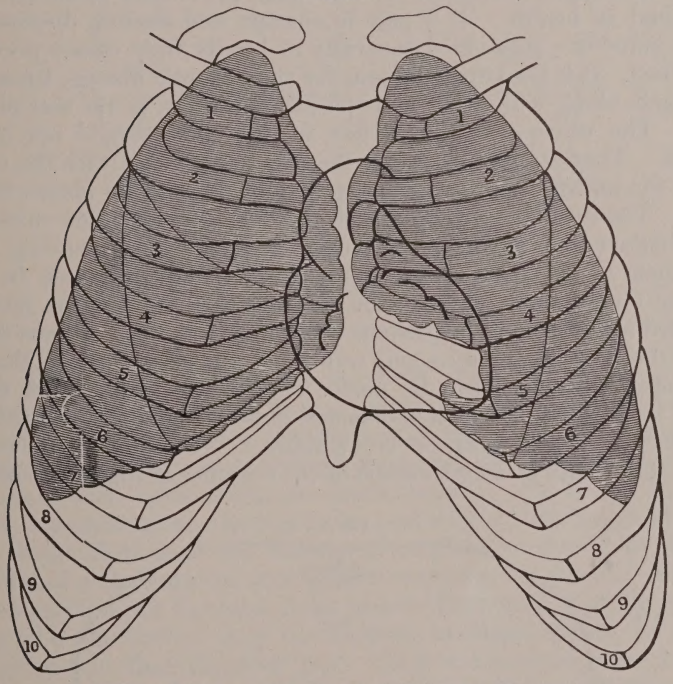
Purulent effusions in the left pleural sac frequently point at the fifth interspace beneath the nipple, because this is the weakest point of the chest covering. A little external to the inferior angle of the scapula and the eighth and ninth interspaces a similar weak point is found.

Limits of the Lungs. The apices of the lungs reach three to seven centimetres (one and one-fifth to two and three-quarters inches) above the clavicles in front; behind they rise as high as a line drawn transversely through the spinous process of the seventh cervical vertebra. The lower margin of the right lung, when the chest is passive, commences at the insertion of the sixth rib with the sternum, and runs parallel with the upper border of the sixth rib to the axillary line. At this point it descends to the upper margin of the seventh rib. On the left side the lower limit extends as far downward as the right. Posteriorily, both lungs reach to the tenth rib. With full inspiration the lungs descend both in front and behind almost the extent of one interspace, while in deepest expiration they are elevated almost to the original position. The "complemental space" of Gerhardt is the space at the lower margin of the lung and at the point at which the left lung overlaps the heart, in which,

during expiration, the surfaces of the visceral and parietal pleura come together. In inspiration the thin layer of lung in both situations is insinuated into this space. The heart interferes with the extension of the left lung. The figure shows the relationship to the chest wall. The space is triangular in shape, extending in the median line from the fourth to the sixth rib. The left edge of the triangular area corresponds to the edge of the left lung, which, notched for the heart, diverges from the median line and runs along the cartilage of the fourth rib.

Position of the Lobes. The accompanying diagram illustrates the position of the lobes of the lungs anteriorly. In the right lung the upper lobe in front extends to the fourth rib, in inspiration laterally to

FIG. 32.



Outline of the heart, its valves, and the lungs. (HOLDEN.)

the third, and behind to the spine of the scapula. The lower lobe begins with the spine of the scapula and extends to the tenth rib behind, and from the fourth to the tenth ribs when fully expanded in the axillary region. The middle lobe is not seen behind; it extends between the third and fourth ribs in the axillary region in inspiration. In front it extends from the lower margin of the upper lobe to the sixth rib.

The upper lobe of the left lung extends to the sixth rib in front and to the fourth interspace at the side. Behind, a small portion extends above the spine of the scapula, while the lower lobe extends from the spine of the scapula to the base of the lung behind. At the sides it

extends from the lowest limit of the upper lobe to the level of the eighth rib.

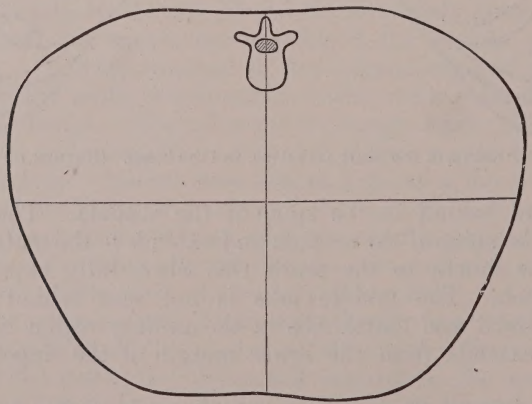
Inspection. By inspection we learn (1) the appearance of the external surface, (2) the shape and size, and (3) the movements of the chest. The second indicates the capacity of the lungs; the last, the degree of functional activity.

Methods. The patient must be seated, if possible, in an easy position, with the light falling directly on the part or from the side. He should be viewed by the observer standing first in front, then behind, and also laterally. The arms should fall by the side; the breathing should be quiet and undisturbed by talking or unusual movements.

The Skin and Subcutaneous Tissue. In health the normal covering should be supple, elastic, or of the color previously described of an individual in health. It is pale in anæmia and wasting diseases; yellow in jaundice; pigmented generally or locally from causes previously mentioned. It is the particular seat for the parasitic disease, tinea versicolor, and, along with other non-specific eruptions, is the seat of sudamina. The veins over the surface of the chest should not be very distinct. They are distinct when there is interference with the circulation in the mediastinum by aneurism or morbid growths obstructing the veins. The capillaries along the base of the chest are often enlarged or more distinct than usual and arranged in a bow corresponding to the attachment of the diaphragm. This bow is frequently seen in intrathoracic obstruction. Œdema or subcutaneous emphysema occurs as indicated under general inspection. If there is too much fat over the surface of the chest, the muscles may want tone, and an estimation, therefore, of respiratory capacity can be made. Wasting of the fat and muscles is seen in phthisis, carcinoma, diabetes, muscular atrophy and paralysis.

The Shape and Size of the Chest. We appreciate the *shape* of the chest in health by an estimation of the relations of the antero-posterior

Fig. 33.

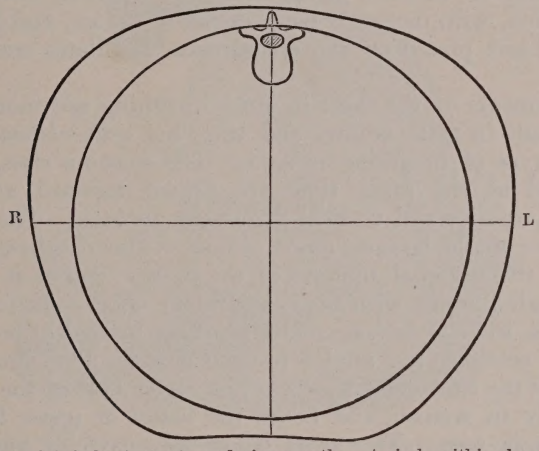


Transverse section of healthy adult chest upon level of sterno-xiphoid articulation. Circumference = 89 centimetres. (Dr. Gee.)

and the transverse diameters and by the shape of the transverse section of the chest. The latter is an ellipse, and has been described as reniform

(see Fig. 33). The antero-posterior diameter is about one-third less than the transverse. Measurement with the cyrtometer (see Mensuration) verifies the result of inspection with mathematical precision. In children a transverse section is different. It is more circular, and the antero-posterior and transverse diameters are almost equal. (See Fig. 34.) Marked deviations from such section, or in the relations of the diameters, are seen in abnormal types of chest.

FIG. 34.



Transverse section of an infant's chest, aged nine months. A circle within shows the similarity.

It is difficult to describe the *shape* of the chest in health. By repeated practice we readily form a judgment of the true shape. No rule has been applied to the relationship of the length of the chest to the length of the body, but it would seem that the circumference of the chest bears such relationship (see Mensuration). In health the chest should be symmetrical, the right side probably a little larger than the left. In the ideal chest the muscles of respiration should be well developed and a moderate amount of subcutaneous fat found. The sternum should project forward from above downward, and the portion joining the manubrium and the xiphoid cartilage should be a little more prominent than the other part. It is not unusual to see a clearly marked distinction between the upper and middle portions of the sternum, or an undue projection of one or more of the upper ribs, and some striking changes about the xiphoid cartilage, none of which are indications of disease. The xiphoid may be depressed, on account of which a crater form or funnel-shaped depression is seen (occupation). The tip of the cartilage is sometimes drawn inward, but more frequently the reverse is noted.

The Movements of the Chest. The frequency, the rhythm, and the degree of expansion, are studied. A complete respiratory act consists of two events, inspiration and expiration. Inspiration is active; expiration, passive. The latter is a trifle longer than the former, as may be illustrated by the following proportion—Insp. : Exp. : : 5 : 6. A pause follows the act of expiration. The chest increases in circumference

and in vertical length (descent of diaphragm) in inspiration as the lung expands with air. The term *expansion* is applied to the act of inspiration; its degree varies.

The frequency and character of the *movements* in health vary in the two sexes. The respirations are from 16 to 24 in the minute in a healthy adult. In the female they may be 20 to 22. In children the frequency of respiration is much greater: under one year 44 per minute, and at five years 26. They are *increased* in frequency in the standing position. They are lessened in the horizontal position, increased during bodily exertion, with increased temperature of the air, and during digestion. The hand placed on the epigastrium facilitates counting of the respirations.

The movements of the chest in quiet breathing are more marked in the lower half in male adults, and thus the *costo-abdominal* or *diaphragmatic* type of breathing is seen. The sternum rises, the ribs are elevated and at the same time are drawn forward and outward. The antero-posterior and vertical diameters increase. The costal angle and epigastric angle become more obtuse. The diaphragm acts conjointly with the external muscles of the thorax, and as it descends the epigastric region swells with each inspiratory effort. In expiration the sternum falls, the ribs become more slanting instead of horizontal, the epigastrium retracts, the angles become acute. The antero-posterior diameter and the transverse lessen. The upper half of the chest moves more actively in women, and hence the *costal* or *upper thoracic* type of breathing is seen. The areas below the clavicles and the upper portion above the sternum swell more distinctly during inspiration. The movements of the lower portion, and especially of the diaphragm, are limited.

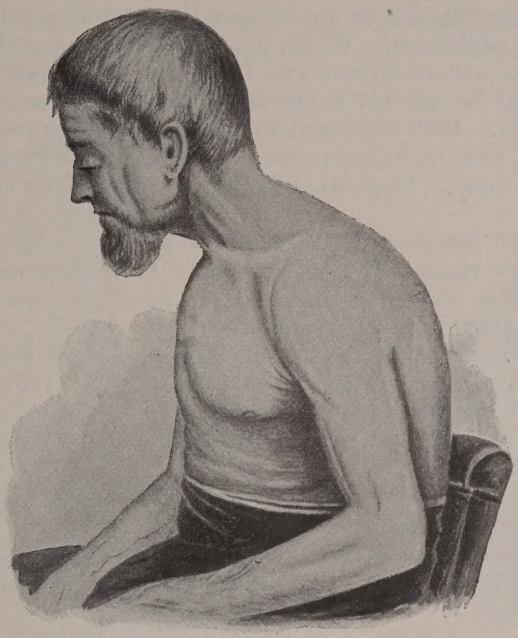
The *costal* type occurs most frequently in children. The type of breathing is *costal* in both sexes during sleep; the same type is observed during deep respiration.

The Shape and Size of the Chest in Disease. Enlargement or diminution may be seen. Such change may be general or bilateral, unilateral, or local.

1. General or Bilateral Changes in Shape. *Enlargement.* The "barrel-shaped" chest, the type of bilateral enlargement of the chest, is seen in health when it is in the state of full inspiration. All the diameters are increased, particularly the antero-posterior; the length is shortened. A transverse section approaches a circle. (See Figs. 35 and 36.) The diameters are almost equal. The ribs are elevated and almost horizontal, the epigastric angle is obtuse. The shoulders are rounded and elevated, and the scapulæ lie flat against the thorax. All the muscles of respiration stand out prominently, the neck and upper trunk muscles particularly. The individual with bilateral enlargement of the chest presents a striking appearance. The neck is short, the arms are short; there is undue fulness above the clavicles. As this enlargement is attended with dyspnœa, the face is drawn and anxious, and the lips usually faintly livid, or purple.

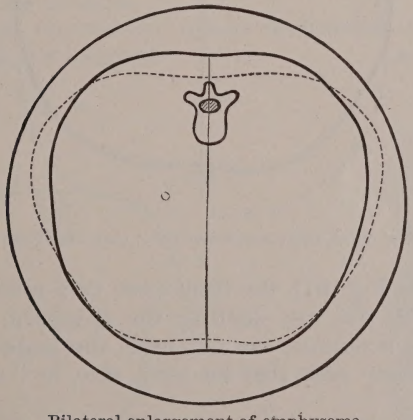
The *movement* of the chest in bilateral enlargement. Expansion is lessened. The respiratory capacity is diminished. The chest is in a state

FIG. 35.



Emphysema with enlargement of the chest. The antero-posterior diameter is much increased. (From PAGE.)

FIG. 36.



Bilateral enlargement of emphysema.

Inner line = emphysematous chest.
Outer line = a circle drawn to show how nearly the emphysematous approaches the circular shape.
Dotted line = natural adult chest.

Actual measurement in centimetres.

Circumference	= natural	89.	emphysematous	87.75.
Transverse . .	= "	29.6	"	27.25.
Antero-posterior	= "	22.25	"	25.4.

(DR. GEE.)

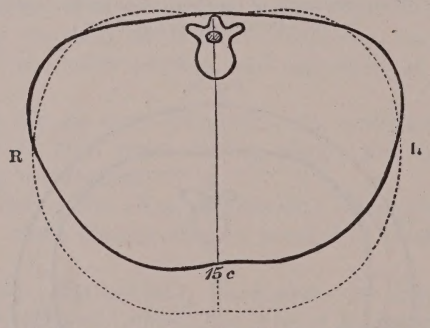
of full inspiration, and the attendant dyspnœa is known as expiratory dyspnœa. The respirations are hurried, the inspirations short, followed by prolonged expiration. While the expansion of the chest in health extends over an area of three or four inches, when the chest is bilaterally enlarged it may be lessened to one and a half inches, or even be as low as half an inch. Both the costal and diaphragmatic types of breathing are seen in a state of exaggeration. In men the diaphragm acts very vigorously at times. Expiration is three or four times as long as inspiration.

Cause. The increase in size arises because of enlargement of the contents of the chest. The increase may be from excess of normal contents or from abnormal contents. In nearly all cases it is due to an increased amount of air within the thorax (normal contents), as in *emphysema.* In a few instances enlargement of both sides is seen in cases of *bilateral pleural effusion,* but as considerable effusion would be incompatible with life, the enlargement from this cause is never very great. It is said that in rapidly growing *cancer* of the *lungs,* such enlargement may occur.

It must be remembered that emphysema can exist without bilateral enlargement of the chest.

Bilateral Diminution in Size. The type is seen in so-called phthisical or tuberculous chest. The chest is long, the antero-posterior

FIG. 37.



The flat or phthisical chest, short antero-posterior, long transverse diameter. (GEE.)

diameter small (see Fig. 37), the transverse very much increased. The angles are acute, the ribs are slanting, the epigastric angle is particularly sharp. The shoulders are not high, the scapulæ are prominent —so marked in many cases that the term alar, or "winged" chest has been applied to it.

Associated with this type of chest the neck is long, the larynx (Adam's apple) very prominent, the arms are long. The patient is loosely put together; the length of the long bones is increased.

It is known as the phthisical, phthisinoid, or tuberculous chest (see Figs. 38 and 39). Although the term tuberculous is applied to the chest of this description, it does not necessarily imply that an individual with such a chest has, or will have, tuberculosis. It is true that in individuals with such type of chest the vulnerability to the

action of the tubercle bacillus is more marked, and they are more liable to have the disease. Nevertheless a very large number of individuals go through life with such chests and die of other diseases. As long as

FIG. 38.

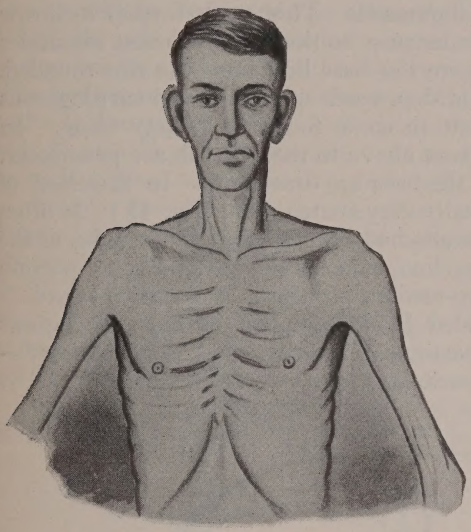
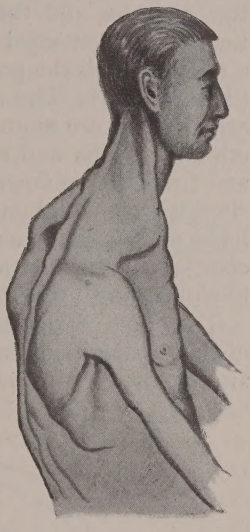


FIG. 39.

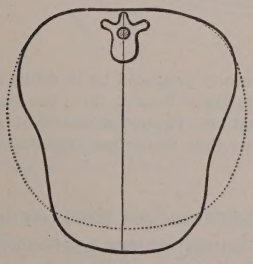


Phthisical, phthisinoid, or tuberculous chest. (EICHHORST.)

they are not exposed to the exciting cause of the disease they most surely will escape its ravages.

Cause. Bilateral diminution means diminution of contents. The extent of air-surface is lessened.

FIG. 40.



Circumference = 42.75 centimetres.
Rickety chest. Dotted line indicates the shape of chest in an infant about the same age. (GEE.)

FIG. 41.

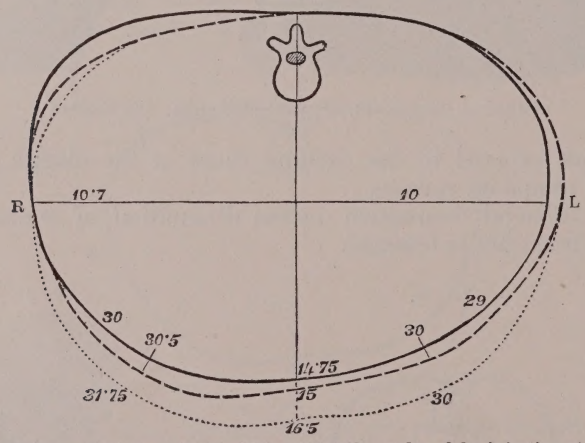
Chest of rhachitis.
(EICHHORST.)

The Chest of Rhachitis. Another type of diminished chest is constantly referred to. It is known as the chest of rhachitis (see Fig. 40), and arises in infancy on account of this disease of the bones. Many

other shapes are seen to which various names have been given. Among the more common is that which causes the "pigeon-breast." (See Rhachitis, and The Head.) The chest is usually shortened, the sternum is much more prominent than in health, the lower portion projecting to an unusual degree. The portion of the chest at the junction of the cartilages and the ribs is depressed. This tends further to throw the sternum outward. The transverse section of such chest resembles a triangle with the portions where the base line joins the ribs rounded. (See Fig. 40.) The sternum is depressed and the osteo-cartilaginous articulations are more prominent in some forms of rickety chest. In others the ribs and sternum from above to the fifth rib are prominent, and from thence downward to the base are drawn in. In the chest of rhachitis the costal angle is usually very acute. (See Fig. 41.) It often looks as if pressure as by the hands had been applied to the sides of the chest about the anterior axillary line, causing the antero-lateral portion to sink inward, while the antero-median portion is projected forward.

The chest of rickets is attended by enlargement of the articulations of the cartilaginous and bony portions of the rib—the rhachitic rosary—and by changes in the other bones.

FIG. 42.



Unilateral enlargement of chest (right side), artificially produced by injecting air into the right pleural cavity. Unbroken line: outline before injection. Broken line: outline after moderate distention. Dotted line: outline after extreme distention. Figures at bottom of vertical line indicate the antero-posterior diameter; along horizontal line, transverse semi-diameter; remaining figures, right and left semi-circumferences. (GEE.)

The *rhachitic* chest must not be confounded with such changes in shape due to abnormal conditions of the upper respiratory apparatus in early childhood. In cases of *adenoid disease* of the pharynx (see Diseases of the Pharynx), the change in shape of the chest has been noted. The *transverse groove* is also seen in addition to the projection of the sternum forward and the lateral grooves along its borders. This extends from the median line along the base of the thorax, corresponding with the junction of the diaphragm with it. It may mark the upper limit of the liver on the right side as it occurred in infancy.

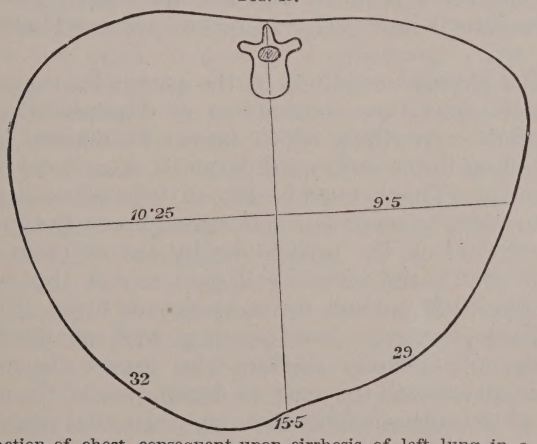
The shape of the chest just described (rhachitic) does not indicate any disease of the lungs; it does indicate deficient respiratory capacity, and of course is the tell-tale by which rhachitis in early life or early laryngeal and nasal obstruction are recognized.

Unilateral Changes in Shape. *Unilateral Enlargement.* This can usually be seen more prominently at the base. The ribs are elevated, the side more rounded, the costal angle more obtuse. The interspaces are frequently effaced, or fuller than on the corresponding side. The *movement* may be increased or diminished, depending upon the cause. The nipple is displaced outward. The scapula of the affected side is also displaced outward, and hence the distance from it to the spine is greater than on the opposite side. (See Fig. 42.)

Cause. Enlargement of one side means enlargement of contents. It may be due (1) to increase of the normal contents, as in compensatory emphysema, in which there is an increased amount of air in the lung, or (2) the addition of abnormal contents, as fluid or air in the pleural sac. It is the most characteristic sign of pleural effusion. When the normal contents are increased the movement is increased; when the pleural cavity is filled it is diminished.

Unilateral Contraction or Diminution in Size. The costal angles are sharper, the plane of the anterior or posterior portion, or of both, is

Fig. 43.



Unilateral retraction of chest, consequent upon cirrhosis of left lung in a girl of fourteen years. The figures indicate antero-posterior and transverse diameters and semi-circumferences of right and left half of chest. (Gee.)

depressed, and approaches the transverse median plane of the chest (see Fig. 43). The semi-circumference is lessened, and the diameter through the nipple or any fixed point is lessened. The interspaces are lessened in width and may be drawn in. The ribs are closer together, and may almost overlap. The *movement* of the side is lessened.

Cause. Any diminution of contents will cause diminution of the affected side. This may occur from obstruction or compression of the bronchi of that side lessening the amount of air in that portion of the thorax. Theoretically it may occur in any case where there is

complete occlusion of the main bronchus. The condition is rare, and is accompanied by marked associate emphysema of the other lung. The unilateral change is most frequently seen in cases of chronic pleurisy. A large portion or even the whole of the lung may be bound down and compressed by thickened adhesion. The pleural cavity of the side thus affected, save where encroached upon by the heart or by invasion of an emphysematous portion of the lung of the corresponding side, is completely obliterated.

Local Changes in Size and Shape. *Enlargement* and *diminution* are also seen.

Local Enlargement is particularly noted in the region of the heart and great vessels, and will be considered when this division of the subject is discussed. A local enlargement in the lower anterior or lateral region of the chest may occur in cases of empyema, in which the pus tends to be evacuated, or in pulsating pleurisy. Enlargement in diseases of the mediastinum is usually seen in the region of the heart and vessels, to which reference must also be made.

Local Contraction. This may be seen either at the apex or the base. At the apex the local contraction or diminution in size is seen above and below the clavicle. The term *flattening* is applied to this condition. The interspace is sunken and the ribs depressed. It may be more readily seen when looked at from behind. Flattening may be either in the lateral or posterior region at the base. The anterior and lateral, or the lateral and posterior region, are combined in the local contraction.

Cause. The physical condition of the part is the same as in unilateral or general contraction—contraction or diminution in size of the parts underneath. Anything which lessens the amount of air in the area corresponding to the contracted part will cause local diminution in size, or *flattening.* This is notably seen in *tuberculosis,* in which affection three processes, alone or in combination, lessen the amount of air: First, the occlusion of the bronchioles by the eruption of tubercles, on account of which the alveoli collapse; second, the overgrowth of connective tissue which attends the more chronic forms of tuberculosis; third, a localized pleurisy. *Local pleurisy,* with organization and contraction of the inflammatory exudate, also causes diminution of the amount of air underneath the part, or diminution of the contents from compression of the adjacent lung structure. In local contractions there is generally diminished movement of the part.

General Review. It is not to be forgotten that in all these changes in shape and size of the chest, with the exception of unilateral enlargement, the element of time is necessary to produce them. In emphysema the change in shape develops over a considerable period. The unilateral and local contractions just spoken of also make slow progress, and hence require a more or less chronic disease for their development. The occurrence of pleural effusion may cause unilateral enlargement very rapidly.

The Movements of the Chest in Disease. *Bilateral Changes. Frequency.* The movements are *increased* in nearly all forms of dyspnœa. (See Dyspnœa.) The frequency of movement varies in many affections. They are more markedly increased in the acute lung affections attended

by fever, and are especially more rapid in children. Increased frequency of respiration does not necessarily indicate pulmonary disease. It always is seen in fever, and is a marked phenomenon of hysteria. Conditions outside of the chest increase the frequency, as enlargement of the abdomen from any cause encroaching upon the capacity of the chest. The respirations are *lessened* in frequency in cases of disease of the medulla in which there is pressure upon the respiratory centre, and in some forms of poisoning, as that due to opium.

Alterations in the Rhythm of Movement. Alterations in the character and *rhythm* of the movement are observed by inspection. (See Dyspnœa.) The movements may be (1) slow, and either shallow or deep; (2) rapid and shallow or deep; (3) irregular in rhythm. The relation of inspiration to expiration in health is as 5 to 6; in women, children, and the aged, 6 to 8. The expiration is longer. The expiration may be prolonged, so that it is far greater in length than inspiration. *Length of inspiration increased.* The degree of expansion and the duration of inspiration are increased when there is obstruction in the trachea or larynx. Such increased expansion is usually associated with retraction of the soft parts of the thorax, especially at the base. The ribs and the tissues along the margins of the thorax are drawn in with each act of inspiration. The space occupied by the lung above the clavicle may also be retracted. The transverse groove is more pronounced. If the difficulty of breathing continues, the indrawing becomes very marked, and, if the ribs are soft, permanent. *Expiration prolonged.* Inspiration is short and quick in cases of emphysema. The *expiration* is correspondingly prolonged, and the muscles of expiration are seen to be brought into full action; the act extends over a long period of time.

In the consideration of dyspnœa the appearances will be described, the action of the muscles of respiration noted, and the position that the patient assumes detailed. (See Subjective Symptoms.)

Irregular Rhythm. By inspection the Cheyne-Stokes type of breathing can be noted. "Respiratory pauses" of half to three-quarters of a minute alternate with a short period of increased activity, and during this time twenty to thirty respirations occur. The respirations constituting this series are shallow at first, but gradually they become deeper and more dyspnœic, and finally become shallow or superficial again. The acts of respiration are carried on by an alternation of pauses and groups of modified breathing. Sometimes consciousness is abolished during the pause. Often the pupils are contracted and inactive. When the respirations begin they dilate.

Unilateral Changes in Movement. Increased movement of one side is seen when the lung of that side is acting vigorously from compensation, the other lung being disabled by disease. The whole side moves more rapidly and vigorously. The increased movement is associated with enlargement of the affected side and hyper-resonance on percussion. Unilateral *diminution in movement* occurs when there is diminution of the respiratory surface or occlusion of the bronchial tubes, or from causes outside of the lung. The air-space is lessened in cases of pneumonia, tuberculosis, or any affection which fills bronchioles and alveoli with inflammatory exudation or fluid. The air-space is particularly

lessened by the compression of effusions in the pleura, or of contracted and thickened masses. Occlusion of the bronchus with diminution of the movement of the corresponding side is seen in rare cases in which a foreign body fills the lumen of the tube, or in more common cases of pressure externally upon the bronchus by an aneurism or mediastinal tumor. Outside of the lung lessened movement is caused by (1) interference with the muscular activity of that side from rheumatism of the intercostal or respiratory muscles; (2) pain seated either in the ribs or in the pleura.

It may be due to *acute pleurisy*, the patient checking motion of the affected side as much as possible, and breathing with the abdominal muscles, because chest respiration causes acute pain. Impaired motion from this cause or from *pleurodynia* may be suspected when it has come on suddenly, and when respiration causes acute suffering, usually depicted in the face. Pleurodynia and pleurisy are to be distinguished from each other by the presence in the one case of tender muscles, a more constant and less stabbing pain, and absence of fever, cough, and râles; and, in the case of pleurisy, by the occurrence of stabbing pain in respiration, absence of local tenderness, and presence of fine, dry, or coarse râles on inspiration, with cough and fever.

Impaired motion due to *pleural effusion* is almost always unilateral, develops gradually, following an attack of acute pleurisy, is unattended by pain on respiration, but is attended frequently by great embarrassment of the respiration, and sometimes by orthopnœa. Fever is usually moderate in uncomplicated cases. It is to be recognized by the clinical signs mentioned and by the physical signs of fluid in the pleura.

Impaired motion from *chronic pleurisy* is of long standing and gradual development. The chest wall upon the affected side is retracted, and may be very markedly sunken. In the absence of accompanying lung trouble there is no pain and no fever. It is to be distinguished from other causes of impaired motion by the sinking in of the affected side, in sharp contrast with the hypertrophy of the other side; by the absence of fever and pain; by its chronicity; and by the physical signs of thickened pleura and compressed lung. Impaired motion from *pneumothorax* develops suddenly, generally in a person with tuberculosis of the lungs. Its appearance is usually precipitated by coughing, and its sudden development is marked by intense pain, distention of the affected side, great difficulty in breathing, and a very anxious expression of countenance. The escape of air into the pleural cavity is followed by the development of pleurisy with effusion, so that the affection presents the physical signs of air and fluid in the pleural cavity.

Impaired motion from *pressure* on a bronchus by an aneurism or enlarged lymph gland produces the physical signs of collapse of the lung coupled with those peculiar to the cause of the occlusion of the bronchus. It develops gradually, the patient having no pain in the lung.

The motion of the affected side is sometimes impaired in *pneumonia*, when a large portion or the whole of one lung is involved, and the air-vesicles are so occluded that very little air can get in. The physical signs in these cases resemble those of pleurisy with effusion very closely; but the diagnosis can be made by noting the acute onset of the disease,

with high temperature and frequent respiration, without antecedent pleurisy, and by the presence of cough with expectoration containing the pneumococcus.

Local diminution of the movement or deficient expansion occurs under the same circumstances in which we find flattened and local contraction, and for the same reason. Hence, in the early stages of phthisis, or in local pleurisies, deficient expansion is observed.

Impaired motion, due to consolidation of the lung in tuberculosis, is usually limited to one or other apex, and is accompanied by flattening of the affected apex and emaciation. The condition is of gradual development, and presents the usual signs of tubercular consolidation of the lungs (*q. v.*)

Sometimes the impaired motion and flattening are due to a superficial cavity from tuberculosis or abscess, and when the walls are very thin they may be seen to flap feebly with respiration.

Rarer causes of impaired motion of the lung are cancer and hydatid cyst (*q. v.*)

PALPATION. By palpation the results of inspection are confirmed, the character and consistence of tumors ascertained, and the vocal fremitus determined.

Method. The surface should be bared, although the fremitus can be detected through a thin layer of linen or gauze. To detect the fremitus in front, it is often well to stand behind the patient, with the palms of the hands placed over the surface of the chest in front. The opposite position is taken to detect the fremitus behind. The axillary region must also be investigated. The hands should be warmed and applied evenly to the surface. The two sides must constantly be compared, either by simultaneous application of the hands on the two sides, or by applying the hand first on one side, then on the other.

Cause. The columns of air in the bronchial tubes are thrown into vibration during the act of speaking. The vibrations are transmitted to the hand on the surface of the chest. They are known as the vocal fremitus. In infants the cry must be relied upon instead of the spoken voice.

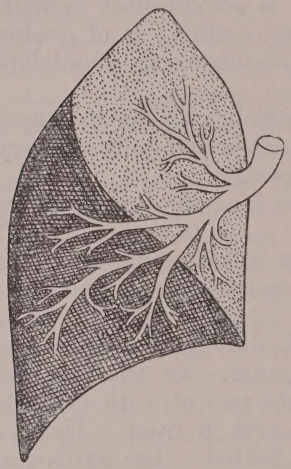
Vocal Fremitus in Health. The fremitus on the right side is stronger than on the left, because the right bronchus is larger than the left, and its angle with the trachea is more acute. The fremitus is stronger in persons with deep voices of low pitch because the vibrations are not so rapid. It is more distinct, therefore, in males than in females, and in individuals with bass voice. The vocal fremitus is felt more distinctly in persons with thin chest-walls. Thick chest-walls and large mammary glands interfere with the transmission of fremitus. The fremitus is not distinct in children because the vibrations are too rapid.

It is well to become familiar with the vibrations produced by fixed monotones in order to appreciate the fremitus. The patient is asked to count one, two, three, or to repeat ninety-nine three or four times. It is well to observe a fixed rule as to the words used, in order to have definitely fixed in the mind the character of the vibrations in health, and the departures from the normal in disease.

Vocal Fremitus in Disease. The vocal fremitus may be increased, may be diminished, or may be absent entirely.

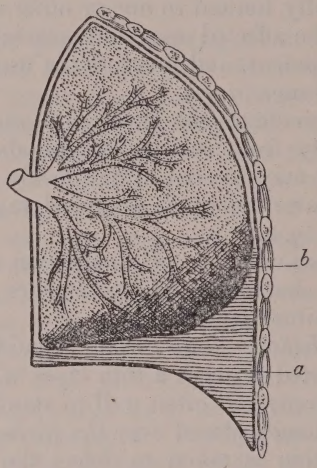
Vocal Fremitus Increased. When the lung is consolidated, vibrations are transmitted with greater force to the hand. Fremitus is increased in all consolidations, as in pneumonia, tuberculosis, and hemorrhagic infarct. (See Fig. 44.) The fremitus may be absent in rare cases of

Fig. 44.



Consolidation: Pneumonia. Vocal fremitus increased. (Gibson and Russell.)

Fig. 45.



Pleural effusion. Vocal fremitus absent at *a*. (Gibson and Russell.)

pneumonia, in which the large tubes are occluded by exudate. The fremitus is increased in the later stages of tuberculosis, when cavities have formed, if the walls are dense.

Vocal Fremitus Diminished. Anything intervening between the lung and the surface of the chest which interferes with the conduction of the vibrations diminishes the fremitus. The fremitus is diminished in cases of thickened pleura and in thin layers of pleural effusion. The fremitus is lessened if the columns of air in the bronchi are smaller on account of diminution in the calibre of the latter, as in bronchitis or in emphysema and asthma. The fremitus is lessened in cavities filled with fluid, or when the bronchus is occluded.

Vocal Fremitus Absent. 1. The vocal fremitus is absent when the columns of air are obstructed entirely by occlusion of the bronchus, as by the external pressure of a tumor, aneurism, or enlarged gland. 2. The fremitus is absent in large accumulation in the pleura of air or of fluid, which is a different conducting medium, causing interference with the vibrations. They are cut off completely, and result in absence of vocal fremitus. (See Fig. 45.) The well-known illustration of striking a stone underneath the surface of the water applies. If the ear of the listener is above the water the sound cannot be heard. If the ear is underneath the water the sound is heard at a long distance from its origin. Vocal fremitus is absent in pneumothorax, in hydrothorax, in pyothorax,

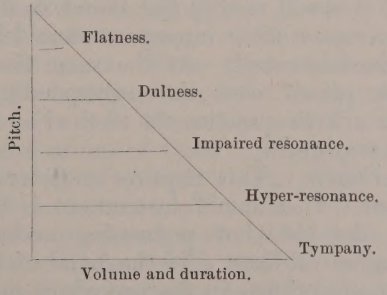
and in hæmothorax. The same physical condition is present when the pleura is greatly thickened, and hence the fremitus is also absent.

The sounds produced by the passage of air through mucus or fluid in the bronchial tubes are transmitted to the hand when it is laid on the surface of the chest. They are known as *rhonchi*. They are felt during inspiration. They may be felt all over the chest in bronchitis, or in asthma, as distinct vibrations, sometimes coarse, or again fine, indicating rapidity of movement. The vibration may be transmitted over a localized area in phthisis, due to air passing through fluid in the cavity. They are distinct in children in cases of bronchitis, and often are the source of much alarm to the parents.

Friction Fremitus. An exudation of lymph on the surface of the pleura often causes a vibration which may be transmitted to the hand. It is known as a friction fremitus, and is felt in inspiration. It is usually felt at the base of the chest, in front, laterally, or posteriorly. It is not modified by coughing, and is increased by full breathing. The rhonchi, on the other hand, are influenced by cough and breathing.

PERCUSSION. By percussion, (1) sounds are elicited, (2) the degree of resistance to the percussing finger estimated. When a part is percussed the sounds produced are noises or tones. If a *tone*, the vibrations are uniform and will vibrate in unison with a tuning-fork; if a *noise*, the vibrations produced are without uniformity. We distinguish the sounds by certain characters. They are the *pitch*, the *volume*, the *duration*, and the *quality* of the sound. The pitch depends upon the rapidity of vibrations, hence the number that occur in a definite period of time. It may therefore be high or low. In sounds that are high in pitch the vibrations are rapid. In sounds that are low in pitch the vibrations are correspondingly slower in the same period of time. The volume or intensity of the sound depends upon the amplitude of the vibrations, and varies directly as the square of the amplitude of vibrations. It is modified by the degree of force used in the production of the sound. "Duration" explains itself. These characteristics bear certain relationships. Sounds that are high in pitch are of diminished volume or intensity and of short duration. The accompanying sketch is diagrammatic of the relation of the characters of the sound. (See Fig. 46.) On the other hand, sounds that are low in pitch have corre-

FIG. 46.



Diagrammatic sketch of the relations of the characters of tone. The perpendicular line represents the pitch. The transverse line the volume and duration.

spondingly greater volume or intensity and longer duration. The three characteristics determine the quality of the sound. The term "clearness" is the quality applied to sounds which are of the character of tones. They are low in pitch, of good volume, and long duration. Sounds that are high in pitch, of small volume, and short duration, are of a *dull* quality. Noises, without pitch, volume, or duration, are absolutely dull or *flat*. The former are indicative of the presence of air; the latter, of the absence of air. The tones, or clear sounds, are naturally produced over structures containing air. Structures in which the relationship of air to solid material varies yield sounds which vary in degree of relationship between clearness and dulness. Resonance and tympany are clear sounds which will be explained later.

Method of Procedure. Due attention should be paid to the presence or absence of tenderness which necessarily modifies the results obtained by this method of exploration. Notwithstanding the presence of a considerable degree of tenderness, definite information can be secured by light percussion. In children percussion should be the final step in the examination.

Immediate Percussion. The chest may be tapped by the finger or hand directly. This was the original method of percussing the chest, but is not now in vogue, except when the clavicles and surface of the sternum are percussed. It was known as the *immediate* method.

Mediate Percussion. The method now employed is that in which a medium is selected to intervene between the chest wall and the instrument used for percussing. This medium is known as a pleximeter. It may be a small plate of ivory of suitable size to place between the ribs, or, better still, the fingers of the hand not used in tapping. The plessor is used to create the sound. It may be a small hammer. The one usually selected is of moderate weight, has a firm, light, slightly flexible handle and metal mallet, the ends of which are tipped with rubber. For purposes of class demonstration a plessor of this character, with an ivory pleximeter, is of value, but for bedside work the fingers of the physician are the best.

The Use of the Pleximeter. The pleximeter must be placed in close apposition to the surface of the chest in performing percussion. If the finger is used as a pleximeter, in percussing the anterior portion of the chest, for instance, it must be placed parallel with the ribs. It must not cross them. If it is not in close apposition to the chest the cushions of air between the two will modify the sound so that accurate data are not obtained. Interspace after interspace should be percussed in this manner from above downward. At the same time, if necesssary, the pleximeter may be placed over the corresponding ribs, but parallel with them. With a little practice the method of applying the pleximeter can soon be acquired.

The Use of the Plessor. This requires considerable practice on the part of the student. If a metal instrument is used care should be taken to acquire the habit of percussing under all circumstances with the same degree of force. If the hand of the operator is employed as a plessor several acts in the procedure must be remembered. It is better to select one finger, and preferably the middle finger of the

hand used. Some operators use more than one finger, but with a little practice a sufficient degree of force can be given with one to elicit the sounds essential for distinction. The finger should be bent at right angles and kept in a fixed position. It must be made to strike the plexímeter directly perpendicularly to its plane. If the blow is given other than at a right angle to the part percussed a true sound cannot be obtained. The blows must be made regularly and the force be even. The character of the part investigated will determine the degree of force that should be used. The force of the blow is to come from the wrist alone. Neither the arm nor the forearm must be employed in its creation. Beginning anteriorly with the supra-clavicular fossæ and proceeding downward an interspace at a time, *comparison* should be made with the other side at each step. The axillary portions, and the posterior portions from supra-spinous fossæ to base, should then be examined in this way. *Recapitulation:* Apply the plexímeter in close apposition to the surface parallel with the ribs or interspaces. Do not apply over rib and interspace at the same time. Strike first with one finger, which is bent at a direct right angle. Let it fall perpendicularly on the plexímeter. Let the blows be of *equal* force and in *rhythmical* succession. Let the force of the blow be created by the wrist. Always compare the two sides of the chest, and first percuss the side presumably normal. The arm certainly, and the forearm as much as compatible with wrist movement, should be kept fixed.

Position of the Patient. The best position is the standing one, with the arms allowed to drop loosely at the sides, the head straight, not thrown back, and the shoulders allowed to fall a little forward if they are inclined to do so. Any position which throws the chest muscles into contraction helps to defeat the object of the examiner who seeks to elicit the chest sounds. In percussing the posterior portions of the chest it is desirable to have the patient stoop forward with arms folded. While this renders the muscles more tense, it is of advantage in exposing a larger portion of the chest.

When the patient is confined to bed he should, if not too ill, be allowed to sit up during percussion, as contact with the bed or with pillows deadens the sounds elicited. This fact should be borne in mind when from any cause it is not desirable to have the patient sit up.

All clothing should be removed, if possible. A thin undershirt may be permitted from motives of delicacy, or parts only of the chest be exposed at one time if there be danger of chill.

The Sounds in Health. Three types of sound may be produced by percussing over the healthy thorax for the purpose of study. 1. Tympany over the trachea. 2. Resonance over the lungs. 3. Dulness over the heart. Modifications of these types represent all sounds produced under every variety of circumstances. They will be considered in the order of their importance. The term *resonance* is applied to the clear sound that is produced over the chest on percussion. It is due to the vibration of the chest walls and of the air in the bronchi. "Pulmonary resonance" is a term also used to indicate the same sound. While as stated above the sound produced is called a tone, yet on

account of the relation of the air to the solid structure of the lung, confined in innumerable sacs, a true tone is not produced, *i. e.*, it cannot be pitched with another tone or made to vibrate in unison with one. For practical purposes, however, the term "tone" may be used convertibly with "clearnesss" and "resonance." Its characteristics cannot be defined accurately, and must be learned by repeated practice.

Modifications in Health. The degree of clearness or resonance differs in various parts of the thorax. It is purer in the upper axillary region, at the angle of the scapula behind, and on the anterior surface of the chest, in the second interspace. It is slightly higher in pitch at the right than at the left apex. It is modified by the condition of the chest walls. Thick chest walls, accumulations of fat, the mammary gland, and the scapulæ impair the resonance and require deep percussion. In persons with thin walls the resonance is clear and more pronounced. The elasticity of the chest walls also modifies it. In the aged it is less clear because of rigid chest walls. In children, in whom the chest walls are elastic, the resonance is much fuller or clearer and approaches more nearly the character of a tone. The sounds vary within certain limits in different individuals with perfectly healthy normal chests, as may be seen from the above. Moreover, a sound normal in one part of the chest may in another part indicate disease. It follows that percussion sounds do not have an absolute value; their significance depends upon the individual and upon the part of the chest examined. The student should learn from the outset to compare the sounds developed by percussion of symmetrical portions of the chest, and thus determine the normal for the individual. Below the third rib on the left side the dulness of the heart destroys the value of comparative percussion. *Significance:* Excess of clearness or resonance or hyper-resonance means excess of air, as in vicarious emphysema. Diminution of clearness means diminution of air—increase of solid structure.

Abnormal changes in resonance caused by disease will be considered further.

Dulness. The sound over the heart is dull and may be useful to compare with dull sounds yielded over areas usually resonant. The character of dulness has been described: it signifies the absence of air.

Tympany. When a single cavity with smooth walls, containing air, is percussed, the sound that is produced is a tone of low pitch, of considerable volume or intensity and of long duration. The term "tympany" is applied to this sound. In health it can be elicited over the trachea, over the stomach when it is free from food, over the large intestine, and at times over the small intestines. In addition to the low pitch and large volume, it possesses a peculiar metallic quality which is characteristic. It is a quality of sound with which the student should become familiar, for variations are characteristic of abnormal physical conditions in the lung and the abdomen. It must be remembered that tympany can be developed normally over the posterior portions of the lungs of infants and children. The relationship of this sound to resonance, or the sound produced on percussing the healthy lung, and to dulness produced over airless structures, may be appreciated by reference to the diagram modified from Gee. (Fig. 46.) In pitch, in volume,

and in duration it is lower than resonance. The latter stands midway between the tympany and the dulness. As intimated previously, all varieties of sounds that may be produced and which occupy positions between the extremes noted in the triangle are dependent entirely upon the relationship of air to solid material. The larger bulk of air yields tympany.

The Pitch. The estimation of the pitch of the sound is of the highest importance. It is one distinctive attribute or characteristic which is of special diagnostic significance as to the physical condition of the part. It requires considerable cultivation by practice to estimate it. Its significance in relation to dulness and tympany have been mentioned. Although a high-pitched sound may be considered a dull sound, this is not necessarily so. A sound of high pitch need not be markedly dull, indeed it may be moderately clear. Under the right clavicle in health the pitch is higher than under the left, but not dull in character.

The student may become familiar with the pitch and with alterations in it by percussing over a portion of the lung clearly resonant, as in the third interspace and thence downward on the right side. As the interspaces in apposition to the liver are reached the pitch changes. The fulness of the sound is lessened; it becomes more shallow. The rapidity of the vibrations can almost be appreciated, and, as they increase, the heightened pitch caused thereby is recognized. This normal increase in pitch is due to a thin layer of lung backed up behind by the solid liver. Change in pitch makes it possible to outline organs and pursue topographical percussion.

The Degree of Resistance. This is estimated by the sense of touch. When organs containing air are percussed the sense of resistance appreciated by the finger that is percussed is small, or, indeed, may be said to be absent entirely. The sensation to the finger is as if the parts underneath bounded away. When there is lessened amount of air, and hence more and more of an approach to solid structure, resistance is appreciated. It is of the greatest importance to carefully educate the finger to an estimation of this sense. Often it may be difficult to determine exactly the pitch. Detection of the presence or absence of solid structure can be materially aided by the sense of resistance.

Superficial and Deep Percussion. In superficial percussion the blows are directed lightly over the part percussed. By this manner the sound yielded by the portion directly underneath the hand is elicited. It is for this reason of advantage in percussing over portions of the lung that are thin. Light percussion is also necessary in children and in patients with sore chest walls. It must be employed if the subject has just had a hemorrhage. In deep percussion the blows are given with great force. It brings out the sound of structures situated deeply in the lung or when overlapped by the edges of the lung. It is therefore necessary in cases of deep-seated consolidation; in cases of aneurism that is covered by lung, in order to define its limits, and particularly in order to determine the true height of the liver and the relative area of dulness of the heart.

Auscultatory or Stethoscopic Percussion is a valuable means of precisely defining the limitation of a dull area, as an aneurism or tumor

within the chest, or of determining the limits of organs even of similar physical structure. The stethoscope is placed over the organ the border of which is to be defined, and percussion is begun some distance from it. It is conducted toward the stethoscope, and very much sooner than by ordinary methods the dull sound of the non-resonant structure is transmitted to the ear. If the tympany of the stomach is to be distinguished from the tympany of the colon, place the stethoscope over either one of the organs. Percuss with the finger-tips directly on the surface by immediate percussion. Begin at the stethoscope, and percuss from it. As soon as the limit of the structure percussed is reached a difference of tone or pitch is observed which cannot be detected by other means. Mediate percussion may also be employed.

Object of Percussion. The object of percussion is to estimate the proportion of air contained in the chest to the solid tissue. We can thus determine (1) the size of the lungs; (2) the presence or absence of abnormal sounds by which the physical condition of the part is ascertained; and (3) the size of the other organs in the thorax (topographical percussion), and in the case of the abdomen the position and size of its organs, and the presence of tumors or other solid structures. The *size* of the lungs. *Increase* in size: The boundaries of the lung have been described previously. If the resonance extends beyond these boundaries it may be said that the lungs are enlarged. This is seen in *emphysema.* The area of resonance in this affection extends beyond the clavicles to a greater height than in health. It encroaches upon, and may cause to disappear entirely, the normal area of cardiac dulness; it extends one and a half to two inches beyond the lower margins in health. The upper border of liver dulness is therefore lower—instead of beginning in the fifth or sixth interspace it begins an inch or two below. *Diminution* in size: Shrinkage of the apices (one or both) takes place in phthisis, hence the resonance of health does not extend as high up in the neck. Shrinkage or contraction may take place along the lateral borders or lower edges on account of phthisis or retracting pleurisy, causing diminution in size of the lung and spurious enlargement of the heart or liver. In diseases below the diaphragm, effusion or large liver, the size varies. The area of the dulness due to the size of the heart and the liver, by which the size of these organs is estimated, are considered under methods of examination of the respective systems.

The Sounds in Disease. It may be said in general that when a sound is produced over the thorax which does not correspond with the normal resonant tone, it indicates an abnormal physical condition, or disease. Difference in the percussion note of two exactly corresponding portions of the chest almost always indicates some abnormality.

Change in tone may be general or local. The areas over both lungs may yield a different percussion note from the normal (bilateral); the change may be limited to one side (unilateral); or it may be found in small areas (local).

Increased Resonance. The resonance may be increased or diminished. When the resonance is *increased* the sound is abnormally clear. If it is fuller and clearer than in health, but does not possess the characteristics of the tympanitic note, it is known as hyper-resonance or exag-

gerated resonance. The physical condition which causes exaggerated or hyper-resonance is increase in the amount of air. This increased amount of air may be general, unilateral, or local. When general (*bilateral*) it gives the characteristic sound heard in emphysema. At the same time dull areas are encroached upon. The heart dulness is effaced, the liver dulness lowered. In this affection the amount of air is so great and the tension of the chest walls so exaggerated that hyper-resonance and sometimes a pure tympanitic sound ("band-box" resonance) is produced over the entire thorax. The same increased resonance may be present in acute miliary tuberculosis. *Unilateral* increase in resonance or tympany occurs when there is an increased amount of air in one lung, on account of compensatory enlargement (vicarious or compensatory emphysema), or on account of an increase of air in the pleura. *Local* increase of resonance occurs when a local area of the lung is acting in a compensatory manner. This is seen in cases of phthisis in which the alveoli or lobules surrounding small areas of consolidation are very distended. The exaggerated note may aid in the recognition of a deep consolidated area. The same note, hyper-resonance, is obtained over a portion of the lung above the line of pleural effusion and above the line of consolidation in pneumonia.

FIG. 47.

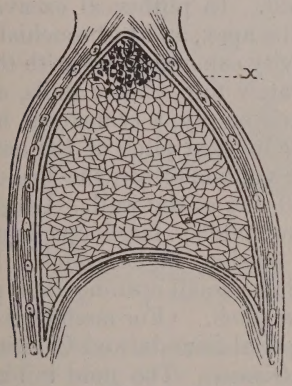


Diagram showing at x moderate dulness over tubercular infiltration. (GIBSON and RUSSELL.)

FIG. 48.

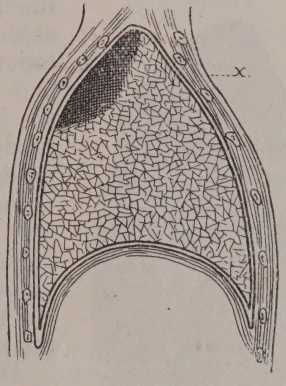


Diagram showing heightening of pitch anteriorly at x from consolidation posteriorly (shaded points). (GIBSON and RUSSELL.)

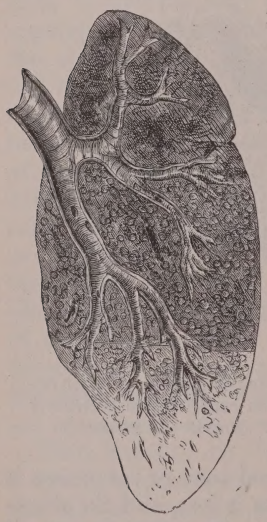
Diminished or Impaired Resonance. The normal tone or resonance is impaired—that is, the pitch is higher, the volume is less, and the duration is shortened—in cases of commencing consolidation of the lung, and in small pleural effusions in which the layer is thin. It is the first change toward dulness, and is particularly noted in the early stages of phthisis. The lung area, usually the apex, is the seat of small areas of tuberculous infiltration. The relative amount of air to solid structure is lessened. Impaired resonance is the result. As the disease advances the note changes gradually to dulness.

Pitch. Gibson and Russell have pointed out the change in quality of sound with change in pitch. (See Fig. 48.) If, for instance, the apex of the lung is percussed in front, and at the same time there is an

effusion of fluid behind, or a consolidation of small area directly on the opposite surface of the lung, the pitch of the sound is raised, when compared with the sound in the opposite lung at the corresponding point. A clear sound of heightened pitch is diagnostic of airless structure behind air-containing structure.

Tympany in Disease. Significance: If a tympanitic note is elicited over a part where in health resonance should be found, it is an indication of disease. It signifies (1) that air is confined in a space (cavity), or an excess of air in many sacs, as the lungs in emphysema; (2) that the tension of the lungs is less than normal—the lung is relaxed, as it is above the limits of a pleural effusion. The issue of a tympanitic sound from the chest occurs—1. As previously stated, *bilaterally*, in cases of emphysema. 2. *Unilaterally*, in cases of pneumothorax and compensatory emphysema. In pneumothorax the pitch may be raised if there is much tension. It is then known as dull tympany. 3. *Locally.* It is limited to the lobe of the lung in some cases of compensatory emphysema. It may occur in the early stage of pneumonia, or in the later stage of complete consolidation. In the former it is due to relaxed tension; in the latter, to the air in the bronchus the lumen of which is free. In cases of pleural effusion, owing to alteration in the tension of the lung, a tympanitic note is present above the layer of fluid. In phthisical excavations at the base or the apex, and in bronchial dilatation, if the cavity communicates with the air, and has moderately thin, elastic walls, and at the same time is empty, a tympanitic note is produced. The musical pitch of the note depends upon the volume of air, the size of the opening, and tension of the wall. Large volume of air, low pitch; large opening, low pitch; greater tension, higher pitch. Small volume, high pitch; small opening, high pitch; less tension, low pitch. (For modifications of tympany see Special Sounds, and Cavities.)

FIG. 49.



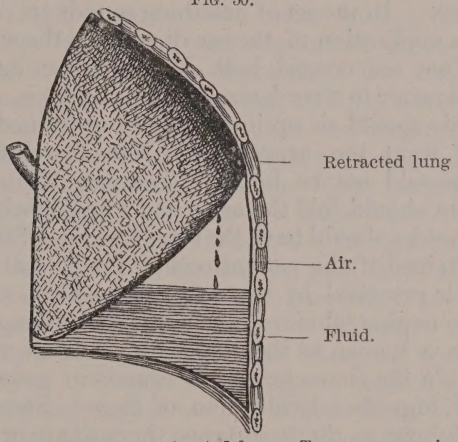
At the apex complete dulness and bronchial breathing, from tuberculous consolidation; in the middle portion impaired resonance, from disseminated tubercles; below exaggerated resonance, from compensatory emphysema.

Dulness in Disease. The note is high in pitch, small in volume, and short in duration. Absence of air, or a relatively small amount in proportion to solid structure, is present. The conditions which give rise to it are all forms of consolidation and pleural effusions. The extent and the degree of dulness depend upon the proportionate amount of solid to air-containing material. Moderate dulness is seen in tubercular disease with moderate infiltration of the lung (see Fig. 47), and in small patches of catarrhal pneumonia, in pulmonary congestion, and in atelectasis and physical conditions in which there is solid material in greater proportion than in health. Absolute dulness occurs when the air is completely absent, as in the stage of hepatization of acute pneumonia, in hemorrhagic infarction, in

condensation from pressure, in pleurisy with large effusion, or great thickness of the pleura, and in tumors. *Flatness* is applied to the extreme degree of dulness. (See Fig. 49.)

We have, therefore, all gradations of the dull sound, from simple impaired resonance in incipient tuberculosis of an apex of the lung, as determined by careful comparison of the two apices, to absolute flatness or deadness. *Method of percussion:* The kind of percussion necessary to bring out the dulness will depend upon its extent and distance from the surface. When the consolidation or thickening is superficial, possibly lying against a thickened pleura, light percussion will discover it, whereas strong percussion would bring out the resonance of the deeper healthy lung tissue to such an extent as to mask completely the superficial dulness. On the other hand, when the airless consolidated tissue is deep-seated and surrounded by healthy lung, strong percussion is required to discover it.

FIG. 50.



Pneumothorax; resonance over retracted lung. Tympany over air. Dulness or flatness over fluid. (GIBSON and RUSSELL.)

Again, when the airless tissue occupies a small focus and is surrounded by healthy lung, as in pneumonia beginning centrally, and when there are small airless foci, as occur sometimes in disseminated tuberculosis, percussion is often wholly negative.

Special Sounds. Special percussion sounds, or sounds the quality of which differs from the ordinary tympanitic sound, are present in some physical conditions. Of these the *amphoric*, or *metallic*, and the *cracked-pot* percussion sounds are most familiar. The amphoric sound is tympanitic, but has a metallic clang, or echo. The prolongation of the sound is compared to an echo. It is like the sonorousness or ring of the voice when one speaks in an empty hall. It can be imitated by percussing an empty vessel. It is heard best in cases of pneumothorax (see Fig. 50) and in phthisical excavation when the cavity is large, is superficial, with smooth walls, and when it has open communication with a bronchus. The cracked-pot sound, as the name indicates, resembles that produced when a cracked vessel is tapped; is it simulated by clasping the hands loosely at right angles to each other and striking

them over the knee. It is heard best over cavities which communicate directly with a bronchus, especially if the chest wall is thin and yields to the percussion stroke. The cavity is usually at the apex. In order to elicit the sound the patient should keep the mouth open. The sound should be created at the time of expiration, and the percussing finger should be retained instead of elevated after striking the plexímeter. In some rare cases this sound can be elicited in health. The other pathological changes with which the sound occurs are in pleurisy above the effusion, pneumonia before consolidation has taken place, and in pneumothorax, if there is a free communication between the cavity and a bronchus. In the latter instance, the sudden rush of air into the bronchus produces this sound. This is proven by the fact that it can be created when the chest is percussed in a case of empyema after the fluid has been evacuated by a free incision. It is to be noted that, while corroborative evidence, it is not alone positive evidence of any single condition.

Auscultation. In the act of breathing sounds are produced. They are heard by the application of the ear directly or through some medium to the chest. They are created both in inspiration and in expiration. They vary in character in accordance with the situation. *Method.* The patient if possible should sit up in an easy unrestrained position. For auscultation in front the arms should hang carelessly by the side. The breathing should not be forced. (See page 258). To auscultate behind the patient should fold the arms and lean slightly forward. For comparison both sides should have the same freedom of movement, which would not be attained if the patient occupied a lateral or side position.

Auscultation is practised by two methods: First, a thin towel free from starch, or a napkin, alone intervening, the ear is applied directly to the chest. This is known as the immediate or direct method. It is of service to ascertain the character of the sounds in general. It has the disadvantage of imperfect localization of them. Second, by means of the instrument known as the stethoscope the mediate or indirect method is practised, but is disadvantageous in infants because the infant cannot be kept quiet or is sensitive to its pressure, and in children because instruments are alarming.

The advantages of the stethoscope over direct methods of auscultation are seen when it is necessary to localize sounds. The definite localized area in which the sound is produced can be ascertained, and sounds in close proximity differentiated. Its use is essential in the study of heart sounds. In addition the operator is more likely to escape from contagious diseases and vermin. Moreover, on the score of delicacy, the stethoscope is preferable.

The stethoscopes that are used are the single and double, and they vary in form with the practice of the operator. It should be an absolute rule with the student that he should become familiar with and use one form of stethoscope alone. The single stethoscope is very good to localize and determine the relation of sounds. It also transmits the shock of an aneurismal vessel or of the heart. The objection to it is that it causes pain if the chest is sore, from the weight of the head, and the pressure of the instrument may modify sounds if bloodvessels are auscultated, or sounds in close proximity to the ear, as a friction. In the use of the

single stethoscope the student should be particular first to see that the portion applied to the chest is perpendicular to the plane of the area over which auscultation is practised. Otherwise slight tilting of the instrument will take place and outside noises be transmitted through the tube. The operator should place himself in an unconstrained position and see that his head is accommodated to the position of the instrument, not the latter to the head. The ear-pieces should fit comfortably. If the parts are covered with hair an extraneous sound from friction is produced. Oil should be applied to allay this. The double stethoscope is the most suitable when the patient is made use of for the instruction of classes. It can even be applied over parts that are quite tender. The rule of application to the chest is the same as that of the single stethoscope. The humming sound in the tube is confusing at first.

The Sounds in Health. If the stethoscope is placed over the trachea at the top of the sternum a sound characterized as follows will be heard: First, it attends inspiration and expiration with a definite pause between; second, the inspiration and expiration are equal in length; third, they are of a soft, blowing character. The inspiration is perhaps a little stronger than the expiration. If the mouth is closed there is no change except that both inspiration and expiration are harsher and sharper. *Bronchial breathing* is the term applied to the sound which is heard in this situation. It is one of the normal sounds of the chest. It may be heard behind, at or a little below the seventh cervical vertebra, feebler in quality than in the trachea, and in the interscapular space over the large bronchi as they leave the trachea. A sound heard in these areas, bronchial in character, is normal.

Vesicular Breathing, or the Respiratory Murmur. If the ear is applied over the anterior portion of the chest, or better still, in the upper axilla or below the angle of the scapula behind, a sound is heard both on inspiration and expiration. It differs from bronchial breathing, however, in that inspiration and expiration are changed in length. The inspiration is one-third longer than expiration. The sound of inspiration is soft, breezy, or sighing in character, increasing in intensity to the end of full inspiration. It is immediately followed by expiration, which diminishes in intensity as the air is expelled, and terminates when two-thirds of the expiratory act is completed. The sounds can be imitated by breathing with the lips in position required to articulate "f" or "v."

Cause of the Sounds. The sound is caused by the passage of air through the nares into the wider pharynx when the mouth is closed. The sounds heard over the bronchi, the terminal bronchioles, and the vesicles are probably created in the upper air-passages and transmitted to the ear through the medium of the bronchi. Bronchial breathing is the sound unmodified, transmitted to the ear, weakened only by its distance from the upper air-passages. The vesicular breath-sound is the same sound modified on account of the intervention of the air vesicles between the ear and the larger bronchi. The sound is thus smothered or dampened down. It was held that part of the sound of vesicular breathing, if not the whole, is due to expansion of the vesicles and rush of air through the bronchioles. The proof, however, seems to be in favor of the first view given, chiefly because, when the vesicular

tissue is removed, as in pneumonia or other consolidation, even far distant from the trachea, bronchial breathing is produced.

Modifications of the Sound in Health. Exaggerated Breath-sounds. Bronchial breathing and vesicular breath-sounds are increased in loudness and sharpness by strong, rapid breathing. In certain places within the bounds of health a sound is heard which partakes of the qualities of both bronchial breathing and the vesicular sound. It is particularly noticed in the inter-scapular region about the level of the spines of the scapula in individuals in whom, in this situation, pure bronchial breathing is not heard. Its characters are, first, soft, blowing inspiration, or loud, harsh inspiration ; second, slightly prolonged expiration, more exaggerated, louder, but not harsher, than in health. The term *broncho-vesicular* is applied to this kind of breathing. It is due to the fact that the sound produced in the larynx is conducted to the ear less dampened down or modified because of the smaller number of air-vesicles which surround the bronchus than are found in the remainder of the lung.

The sounds are increased in children, in whom there is combined greater elasticity of the chest wall and greater friction throughout the smaller bronchi, which are relatively larger. So distinct and characteristic is the sound in children that the term *puerile* respiration is applied to it. The sounds of inspiration and expiration are both intensified or sharper than in health; the latter is relatively prolonged.

Feeble Breath-sounds. The sounds are modified by the condition of the chest walls. If they are thick, or there is an abundance of fat, the sounds are fainter or lessened in intensity. Feeble respiratory power, in wasting and exhausting diseases, causes feeble breath-sounds. The condition of the upper air-passages, even if not pathological, modifies the sounds. If the glottis is small, or there is a disturbed relationship between the nose and pharynx, the sounds will be modified. They are usually weakened.

The Sounds in Disease. Before indicating the sounds which arise from changes in the physical condition of the lung, it may be well to call attention to the confusion that always arises when the student is examining the chest for the first time. The probability is that the coincidence of heart and lung sounds in the chest prevents the detection of the respiratory sounds. If attention is paid to the rhythm, they can be distinctly isolated. At the same time that the student is auscultating the lungs, the hand should be placed on the thorax or the epigastrium and attention fixed upon the two acts of respiration—inspiration and expiration. Before attempting to time the breathing, note the occurrence of each movement, the expansion of inspiration and the contraction of expiration, and then note the character of the sound that is heard in each. By this means the sounds of respiration are accurately ascertained, and confusing extraneous sounds, as from the heart, distinctly eliminated. It is well for the student to bear in mind that sounds heard in the chest, which are departures from the normal sounds, always indicate disease.

Vesicular Breathing Exaggerated. Bilateral. The vesicular breathing or respiratory murmur is increased, first, when there is increase in the force of breathing—when normal respiration is increased and the patient takes full, deep breaths. It is seen in some forms of dyspnœa,

as in the acme of Cheyne-Stokes breathing or in the dyspnœa of diabetic coma. It may be increased or exaggerated in certain forms of bronchitis, particularly when the small tubes are narrowed. *Unilateral* exaggeration or increase of vesicular breathing is heard when the lung is acting vigorously, or in a compensatory manner. The strong inspiration followed by strong and relatively prolonged expiration of an actively moving lung signifies almost certainly disease of the lung of the opposite side. *Local* exaggeration of vesicular breathing, the inspiration harsh, is noted in cases of phthisis in its earliest stages. It should be compared with the sound of the opposite side, when the distinction can easily be ascertained. It is heard over the apex, in pneumonia or pleurisy of the base, and *vice versa.*

Vesicular Breathing Diminished or Absent. Anything which lessens the amount of air supplied to the chest diminishes the vesicular breathing. *Bilateral.* It is, therefore, lessened in cases of occlusion or obstruction of the nares, the pharynx, or the larynx. It is lessened in all cases in which the expansion is interfered with. In feeble persons the respiratory murmur is particularly weak behind. If the muscles of respiration are paralyzed or enfeebled, the murmur is also lessened. If the expansion is interfered with on account of disease of the diaphragm or pressure upward by accumulations in the abdomen, it is weakened. Thickened chest walls that occur from disease, as œdema, weaken the respiratory sound. The vesicular breathing is weakened throughout the entire extent of the lung in emphysema; on account of the enfeeblement of respiratory forces and shortening of the act of inspiration, less air enters the already over-full chest; moreover, in the bronchitis that attends emphysema, the bronchioles are all more or less occluded, and hence the air supply lessened. (See Fig. 35.) *Unilateral* diminution of breath-sounds occurs (1) when there is narrowing of the bronchus as in cases of aneurism or mediastinal tumor; (2) when there is pleural effusion, which (*a*) lessens the amount of air-space by compression of the lung and (*b*) interferes as a different conducting medium. (Fig. 50.) If pain in pleurisy, pleurodynia, or neuralgia is present on one side, the breath-sounds of the affected side will be lessened. Not only in pleural effusions from serum, blood, pus, or air, but also in thickness of the pleura there is weakness or faintness of the respiratory murmur. It should not be forgotten that effusions and thickenings of the pleura rarely take place bilaterally; under these circumstances the breath-sounds would be weakened. The degree of enfeeblement is not so great as it is when effusion is limited to one side. Local diminution of breath-sounds occurs in the early stage of phthisis or in the earliest stage of pneumonia.

It is well for the student to analyze the sounds and attend closely to their character during each event of a respiratory act. Having fixed the attention on respiration, noted its divisions and excluded cardiac rhythm, note (1) the character of the sound in inspiration; (2) the character of the sound in expiration; (3) the relative rhythm or length of the two.

Alteration of the Rhythm. In addition to the character of the breath-sounds, we take cognizance of the rhythm of the sounds. In health the movement of inspiration and that of expiration are almost equal, but, as

previously noted, the sound of inspiration is heard during the entire act, while that of expiration occupies the first third or so of the act. The sound produced during expiration may even be less than half the length of inspiration. The following proportion represents relative length—I : E :: 3 : 1.

Expiration Prolonged. The first notable change in respiration, the vesicular murmur remaining normal, is prolongation. When the expiration is prolonged it equals inspiration, or may even be longer. This is due to difficulty in getting the air out of the chest—expiratory dyspnœa, a physical condition by which the sound of expiration is conducted to the ear. It is prolonged in bilateral broncho-vesicular breathing (*q. v.*). Prolongation of expiration all over the chest is seen in emphysema. The inspiration is short, the expiration prolonged. Although distinct throughout the chest, it is more pronounced above the clavicles and along the free margins of the lung anteriorly. *Local* prolongation of the expiration is of great diagnostic significance when areas of the lung are consolidated in part and the elasticity thereby impaired. The respiratory murmur is harsh, or puerile, or it may be weak. This condition obtains in tuberculosis and is one of the first physical signs of this affection.

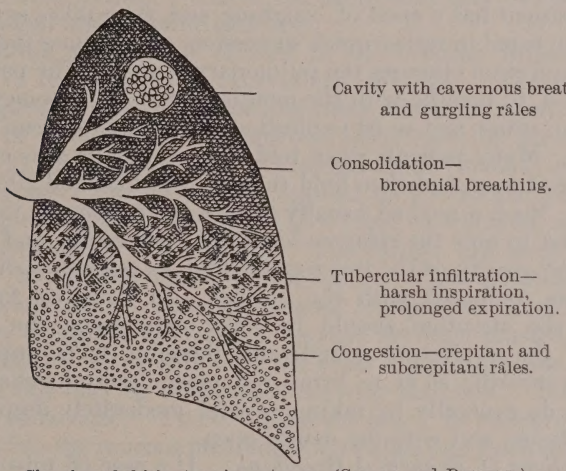
Jerking or Interrupted Inspiration. Instead of the smooth, even, sighing, or breezy inspiration the sound is created in puffs or jerks, so that during the act of inspiration, as the chest expands, a number of successive vesicular sounds are heard until the act is completed. The physical condition which causes jerking inspiration, or cog-wheel breathing, is found in the earlier stages of tuberculosis, when the various bronchioles are more or less occluded by outgrowths of tubercle. The air therefore enters different lobules at different periods of time, and on account of breaks taking place, we have the occurrence of this peculiar irregular sound. It must not be confounded with the same character of breathing that is heard adjacent to the heart, due to the pressure of that organ, or of structures in intimate relation therewith, upon portions of the lung, on account of which air enters various areas in puffs. On the other hand, jerking inspiration sometimes occurs in health. It is heard in nervous patients. While due to the physical conditions mentioned, it is of no significance unless attended by other physical signs.

In cases of adhesions at the apex, particularly of the left lung, the same puffing or jerking inspiration is often heard. It is also present in aneurism, or disease of the aorta, pressing upon a bronchus on account of which the air enters the part in an intermittent manner. When pathological jerking breathing is present the expiration is prolonged, and if the case is under observation a sufficiently long time, bronchial breathing will usually replace the respiratory murmur. Small, moist râles usually attend jerking breathing when it is pathological, especially if excited by coughing or a full breath.

Bronchial Breathing. The normal situation of bronchial breathing in health has been indicated. If the same kind of breathing is heard in any other portion of the lung, it is pathological. It is generally indicative of the presence of consolidation. The spongy lung-tissue is replaced by solid conducting material, by which the bronchial sound

is conducted to the ear. It is heard, therefore, in all pathological conditions in which consolidation takes place. It is the typical form of breathing of pneumonia (see Fig. 51), of consolidation of the lung due to tuberculosis, of hemorrhagic infarcts, and of syphilis. It must not be forgotten, however, that cases of pneumonia do exist without this type of breathing. This is the case when the large bronchus supplying the lungs, or the bronchioles, are occluded by inflammatory exudate. In tuberculous consolidation it may be absent for similar reasons. In central pneumonia, where consolidation is deeply seated and surrounded by lung-tissue, bronchial breathing may not be heard, or it may be postponed until the third or fourth day of the disease, by which time consolidation has reached the surface of the lung. In certain cases of pleurisy with effusion, bronchial breathing exists. The affection is

FIG. 51.


Cavity with cavernous breathing and gurgling râles
Consolidation— bronchial breathing.
Tubercular infiltration— harsh inspiration, prolonged expiration.
Congestion—crepitant and subcrepitant râles.

Showing phthisis at various stages. (GIBSON and RUSSELL.)

not great enough to compress the lung completely. The bronchial tubes remain patent, while the vesicular structure is compressed. A low-pitched bronchial breathing is heard under these circumstances. It is more pronounced at the upper layer of the effusion. It is always heard close to the spine posteriorly, where the lung is compressed. Sometimes it is heard above the limit of the effusion, in all probability because of relaxed tension of the lung.

Varieties of Bronchial Breathing. All their characteristics must be borne in mind. (See p. 253.) It must not be forgotten that bronchial breathing is not represented accurately in every instance by the sounds heard over the trachea. Its character may be modified and yet approach the type of breathing heard at that place. The modification occurs in any one of the two portions that go to make up the sound: (1) The blowing element may not be as distinct in inspiration as in expiration; (2) in rare cases the characteristic blowing sound may not continue as long during expiration as to equal the inspiratory sound.

On the other hand, (3) the bronchial breathing may vary in pitch. At times it is heard in abnormal states (*a*) high in pitch, both in inspiration and expiration, but with a pure blowing quality (harsh) attending each. It may be (*b*) soft and low in pitch attending both acts. The strong, high-pitched sound emitted by breathing deeply when the lips and tongue are placed in position to pronounce "ch" is termed tubular breathing. It is the characteristic sound of croupous pneumonia. (4) The loudness of the sound may also vary. This depends largely upon physical peculiarities of the individual. The condition of the chest walls and the force of breathing determine it.

When pleurisy with effusion coexists with pneumonia, the bronchial breathing, which should be audible, is feeble and distant. Under the same circumstances a bleating sound is heard. (See Ægophony.)

Mode of Determination. Breathing which may, during very quiet respiration, appear to be normal, is sometimes discovered to be bronchial when the patient has a spell of coughing and then takes several deeper breaths than usual in rather quick succession. Sometimes the noise made in nasal respiration obscures the pulmonary sounds. The patient should be instructed to breathe with the mouth open, to take somewhat deeper breaths than usual, and to let expiration follow at once upon the close of inspiration. Many patients when told to take deep breaths expand their lungs to the utmost, and then hold the air in awhile, and allow it to pass out slowly. Such a method usually defeats the purpose of the examiner, which is first to note the relative length of inspiration and expiration, and then the quality of the two sounds, first, as regards each other, and, secondly, as compared with the normal. In listening for bronchial breathing the attention should be fixed more upon the length and quality of the expiratory sound, and therefore it is important that the patient breathe so as to bring out most clearly its characteristics; this he can do generally by taking several moderately deep breaths in quick succession and with the mouth open.

Modifications of Bronchial Breathing. If a case of tuberculous consolidation is watched, it will be found after a time that the bronchial breathing becomes lower in pitch. It is heard in inspiration and expiration, but a more hollow quality attends the sound. From the hollowness of the tone the term *cavernous* has been applied to the breath-sound, and the change in the part that has taken place to cause it is due to obstruction or excavation of the consolidation, or to dilated bronchi. It is a sign of a cavity (see Fig. 51). Cavernous breathing may have a metallic quality attending it, and then it is called *amphoric.* It is analogous to the sound produced by blowing across the open mouth of a jar. A large cavity with smooth walls that communicates with the air is the cause of the development of such sound. It is heard also in pneumothorax, when such communication exists. The metallic tone is analogous to the metallic percussion sound. It occurs under the same physical circumstances. The physical condition which causes it may be so marked that the same character of tone is imparted to râles produced in the cavity, or to the heart sounds which are transmitted by the solidified area surrounding the excavation.

Broncho-vesicular Breathing in Disease. The physical condition is

commencing consolidation surrounded by vesicular structure. It is found midway in the change from respiratory murmur to bronchial breathing in tuberculosis. The inspiration is harsh; the expiration prolonged, harsh, and blowing; or the former may be bronchial or cavernous, the latter absent. It may, however, be indistinct or masked by râles. It is heard sometimes in the earlier stages of pneumonia, and is the modified bronchial breathing which is heard when small areas are consolidated in capillary bronchitis and catarrhal pneumonia, with collapse of lobules. The term "transition breathing" has been applied to this character of breath-sounds.

New Sounds. The foregoing sounds are modifications of the normal sounds that are heard during the act of breathing. New sounds or adventitious sounds are created in the lungs or in the pleura. In the lungs the term râles is applied to them, and in the pleura they are known as friction sounds. Under the same head may be classified the succussion sound and metallic tinkling.

Râles. Râles are sounds created in the bronchi, bronchioles and air-vesicles, or in pathological excavations (cavities). They are due (1) to the passage of air through bronchial tubes which are narrowed, either on account of swelling of the mucous membrane, or on account of spasm; or (2) the passage of air through fluid (mucus, serum, pus, blood). The term "dry râles" is applied to the former class; moist râles, or crepitation, to the latter. Dry râles, or rhonchi, are divided into (*a*) sonorous and (*b*) sibilant. The former are large râles, the character of which is indicated by the name. They are created in the large bronchial tubes. They are coarse, low-pitched musical sounds. The latter are created in the small tubes, and are high-pitched, whistling sounds. Both are heard only over the areas of their creation, although the sonorous râle may be transmitted all over the chest. Both may be heard at the same time. The dry râles are heard in the early stages of bronchitis, when the mucous membrane is swollen and thickened, but has not begun to secrete mucus or muco-purulent matter. They are also heard in asthma in which there is spasm of the bronchial tubes, and in the chronic bronchitis of emphysema. In the latter the smaller râles are more common.

Moist Râles, or Crepitation. They may be divided into large or small râles; the latter are also called subcrepitant. (See Fig. 51.) The *crepitant* râle is a fine râle, said to be created in the alveoli, due to inflation of the cells the walls of which have held together by exudation or fluid (œdema). It is a fine râle distinctly localized, resembling the sound produced by rubbing a lock of hair between the fingers or by putting salt on a hot plate. In the early stage of pneumonia and in œdema of the lungs it is said to be pathognomonic. It, however, may be heard under all circumstances where there is a small amount of fluid in the alveoli and feeble respiratory action. The *small, moist,* or *subcrepitant râles* are created in the smaller bronchioles and the alveoli. They may be general or local. If general, they are due to bronchitis in the second stage. There is an abundance of secretion in the terminal air-passages which is thrown into vibration by the current of air during the act of breathing. The element of moisture is pronounced and gives to them their

character, to which the term "crackling" is sometimes applied. They are found in congestion with outpouring and stagnation of secretion; in œdema; and whenever fluid is drawn into the bronchi, as when there has been a hemorrhage in the upper passages. Small moist râles in local areas are found in phthisis, particularly in the first stage, on account of the local bronchial catarrh, and in the second stage for the same reason. They are also heard in the early stage of pneumonia, particularly in the area of the lung which is the seat of collateral œdema adjacent to the consolidation. They are also heard in the later stages of pneumonia when resolution has taken place. If this is reached, however, they may be replaced by large râles. They may be heard around any consolidation because of congestion, œdema, or catarrh. It must not be forgotten that cough or forced inspiration must be excited before it can be said that râles are absent.

Large moist râles, or *mucous râles*, are created in the larger bronchial tubes, or in cavities, from the same causes that produce them in the smaller tubes. The fluid, however, is larger in amount, the air-current stronger, and the space for vibration is greater. While heard in bronchitis, in their most marked form they are heard in the third stage of phthisis. They are described as bubbling and gurgling râles, and they are very characteristic after a full breath or cough. (See Fig. 51.)

Râles are to be distinguished from other adventitious sounds. Certain characteristics that attend them make this easy, although over and over again it is quite impossible to determine whether fine râles or friction sounds are present. This is particularly the case when the râles are heard over the bases of the lung. We recognize râles, *first*, from the *characters* previously mentioned. *Second*, by their *locality*; if the adventitious sounds are general, they are due to râles. *Third*, râles are *modified* by *cough* or *breathing*. They may be intensified by either act, or, after the completion of the act, may disappear entirely. On quiet breathing, in the early stages of tuberculosis, for instance, they may not be heard at all. It is absolutely necessary before excluding them to have the patient cough and then take a full breath. *Fourth*, they *vary* in *position*. This may occur from hour to hour. If the chest is examined in the morning they may be more pronounced, for instance, at the base. At another time in the twenty-four hours they are distinct at the apex. They are more likely to be present at the base if the patient is kept in the recumbent posture. *Fifth*, they *vary* in *character*. At one time small, moist râles are heard; in a short time they are replaced by larger râles. Of course, the change from dry to moist râles is sure to take place as a pathological condition. In a case of bronchial asthma all sorts of râles may be heard in a few hours. *Sixth*, they seem to be farther away from the listening ear than are friction sounds.

Râles in the bronchi must not be confounded with the crepitant or fine crackling sound which is heard at the base of the lung in patients who have been ill from the exhaustive fevers and who have not taken full breaths for some time. They disappear after the patient has inspired deeply for a half-dozen times.

Râles alone are not diagnostic of any affection save bronchitis, in which, with the absence of other physical signs, their occurrence all

over the chest is significant. In the absence of this affection râles at the bases of both lungs are due to congestion. Râles at one apex, with failing health, point to the possible onset of tuberculosis.

Friction Sound. In health the two surfaces of the pleura rub together without the creation of sound. If they are inflamed, the surfaces are roughened, on account of swelling and dilatation of the capillaries producing a more or less granular surface, or on account of transudation of fluid or lymph. Under these circumstances rubbing together of the two surfaces creates a sound to which the term friction is applied. It is heard at the end of inspiration, and may continue during expiration. It is a localized sound, usually at the seat of pain; it is near to the ear and is not modified by cough or full breathing, except occasionally by the latter when repeated. It occurs in "nests" or "bunches." It may be increased by the pressure of the stethoscope. Moreover, it is a fixed sound, in that it does not disappear until effusion takes place. It reappears again when the fluid subsides. The above characteristics distinguish it from râles. Both, however, may occur together. Although almost always of respiratory rhythm, when the pleurisy is in the neighborhood of the heart the friction may be of cardiac rhythm. Under these circumstances it is more distinct during the act of inspiration. It is heard as a systolic rubbing along the borders of the heart.

We not only distinguish the friction sound by the characters just indicated, but the presence of pain renders its existence more probable. Usually it is heard at the base in the nipple line in front or scapular angle behind, and frequently in the axillary region.

In addition to the friction sound that attends the onset of acute inflammation, creaking sounds of the same nature, not unlike the sounds produced when an old door is swung on rusty hinges, or when new leather is bent, are heard in cases of old pleurisy. Other physical signs of pleural adhesions are present, and often a friction fremitus is transmitted to the hand. An old friction is often heard at the apex, in the neighborhood of old cavities. It attends both inspiration and expiration, is not modified by cough, nor has it any of the elements of moisture that attend moist râles. The patient may be cognizant of the grating or rubbing sensation, and be able to describe this sensation during each breath. It may continue a long time after an acute pleural effusion has been removed, and is sometimes the source of anxiety and inquiry upon the part of the patient.

Pyæmic deposits in the lungs, infarction, bronchiectasis with reactive pneumonia, and pleurisy with emphysema, are first revealed by pleuritic frictions. (Vierordt.) At the base of the right lung they may be the first indication, or an early one, of hepatic abscess. (Clark.) The pleural friction in the hepatic region must not be confounded with peritoneal friction of respiratory rhythm. In secondary cancer of the liver a friction may be heard in the seventh or eighth interspace.

Metallic Tinkling. The idea imparted to the listener is of the falling of some material into fluid in a hollow space. The physical condition is that of a cavity partly filled with fluid, partly with air, into which there is dropping from an opening above. This is seen in hydro- or pyopneumothorax and in a few cases of large cavities. The air-chamber

acts as a consonance-box and resonator, and gives a metallic quality to the sound. Other physical signs of cavity and fluid are associated. It may be heard when the patient is breathing quietly or only after coughing. Sometimes only tinkling is heard, or the sound of a number of drops is transmitted. The latter occurs after coughing takes place.

Bell-tympany. The *bell sound* is heard when air is confined in the pleura. If the stethoscope is placed over the pleural cavity, and two coins are used as plessor and plexímeter, a distinct metallic or anvil-sound is transmitted to the ear. The cavity containing air can be outlined and its extent clearly defined if the metal plexímeter is moved about. As soon as it passes over the surface of the chest underneath which air is not confined the sound is not heard. Although heard in nearly all cases of pneumothorax, there are some cases in which it cannot be elicited, probably because of the size of the aperture in the pleura.

Succussion. The ear is placed to the side of the chest, and the patient's body moved suddenly by himself or by the observer. A splashing sound is heard. It can only be produced when there is air as well as fluid present in a cavity. It was first described by Hippocrates, and the term "Hippocratic succussion" has been given to it. It is characteristic of hydro-pneumothorax, although not present in all cases of this disease. The sound may be audible at a distance. Metallic tinkling can usually be secured at the same time.

Auscultation of the Voice. When the ear or stethoscope is applied to the surface of the chest and the patient asked to speak, the vibrations of the air in the trachea and bronchial tubes produced by this act are transmitted to the chest wall and become audible. It is known as the *vocal resonance.* It is a sign which goes hand-in-hand with *vocal* or *tactile fremitus*, and is modified by the same conditions which modify the latter. In disease it may be *increased* or *diminished.* While, in general, conditions which increase the fremitus increase the vocal resonance also, this is not invariably the case. Sometimes one is increased and not the other, without there being any evident reason for it. It varies in health under similar circumstances. The sound is purring or buzzing. It is heard more pronounced at the right apex than at the left; in persons with thin chest walls; in individuals in whom the voice is low in pitch and strong. It is lessened, therefore, in females and children. It is lessened the farther away the ear gets from the larynx, and hence is feebler at the bases. It is immaterial which words are selected by the patient to create the resonance. It is important for the student, however, to become familiar with the resonance of a definite series of words which when pronounced do not need any marked change in inflection of the voice. The words one, two, three, spoken repeatedly, are selected, or ninety-nine used in succession. The tone of the patient should not be raised or lowered during the act of speaking. Symmetrical portions of the two sides of the chest must be examined successively.

Vocal Resonance Increased. Increased vocal resonance depends upon the intensity or extent of the cause. When slightly above normal it

is referred to as slight increase, or when the voice is transmitted comparatively distinctly to the ear it is known as *bronchophony*. This may be heard in health over the trachea or the bronchi behind. When heard over the vesicular structures of the lung it indicates that the vibrations are transmitted by some better conducting material to the ear. This is usually a consolidated lung, and hence: 1. In all cases of consolidation the resonance is increased, or bronchophony created; but in pneumonia, if the bronchus is occluded by exudate, it is absent. 2. If the lung is collapsed but the bronchi open, the resonance is increased. 3. It is also increased in cavities. Sometimes the resonance is intensified and the sound even more pronounced than when heard over the trachea.

Pectoriloquy. The voice may be so distinctly transmitted that we have the impression that the patient is speaking into the mouth of the stethoscope. If the patient speaks slowly the words may be clearly perceived. It is more striking when the patient whispers. The term "whispering pectoriloquy" is then applied to it. It is detected over a cavity if it communicates with a large bronchus, and in consolidation of the lung.

Vocal Resonance Diminished. Vocal resonance is diminished or absent when anything cuts off the supply of air and intercepts the vibrations from the part over which the observer is auscultating. Fremitus and resonance are absent over the affected bronchial area which is occluded by external pressure, as from an aneurism. Diminution or absence of vocal resonance is more marked in cases of pleural effusion (serum, blood, pus, or air) or thickened pleura. The vibrations are impeded because of the difference of conducting material. The degree of diminution depends upon the amount of effusion.

Modifications of Vocal Resonance. 1. At the uppermost limit of pleural effusions, at which point the layer of fluid is thin, the resonance is transmitted in a modified form. It is tremulous and bleating in character, and because it resembles the sound of a goat is known as *ægophony*. It is especially heard at the angle of the scapula, or below it in cases of moderate effusion. It is due to the fact that the fundamental tones are intercepted by the fluid while the other tones are allowed to pass through and give the peculiar bleating sound. (Gee.) 2. The vocal resonance may have a metallic character in cases of pneumothorax when there is free communication with the bronchus.

Cavities. Pulmonary cavities are due to destruction of lung by abscess, gangrene, or tuberculosis, or to dilatation of the bronchi.

As there is usually a local increase in the amount of air in cavities, there is in consequence a local area of exaggerated resonance, or tympany, and with it the occurrence of cavernous breathing, or breathing of an amphoric type. The presence of a cavity, however, is often difficult to recognize, because of the relation to the surrounding structure or because of fluid contents. If the lung about it is the seat of consolidation the physical signs of this consolidation may override the signs of a cavity. If, on the other hand, compensatory emphysema surrounds the cavity its presence may be scarcely recognized. Moreover, the contents of the cavity render the recognition of its presence diffi-

cult. If it contains a considerable amount of fluid the signs of consolidation alone may be yielded. Much attention has been paid to the recognition of cavities, and some methods employed by which it is thought they can always be distinguished. While it is a satisfaction to determine exactly the presence and location of a cavity, it is not an essential to diagnosis. To be able to confirm the presence of an excavation, even if the physical signs point to its occurrence, the judgment should be controlled by examination of the sputum. If on such examination yellow elastic tissue is found, the presence of a cavity is authenticated. The methods employed to determine their presence absolutely have been named after observers who have devised them.

First, *Wintrich's change of sound.* If the cavity communicates with a large column of air in the bronchus and percussion is employed with a moderate degree of force, the note will change as the patient alternately opens and closes the mouth. If the mouth is open wide the sound is louder and more distinctly tympanitic and higher in pitch. If the mouth is closed the sound is correspondingly lessened and not so tympanitic. Indeed sometimes a sound is obtained with scarcely a trace of tympany. This change of sound is in all probability due to change in the resonant cavities in the upper respiratory tract. It must not be confounded with "Williams' tracheal tone," which can be elicited near the junction of the clavicle and sternum on the left side in cases of consolidation of the underlying portion of the lung, particularly if the force of the blow is directed toward the trachea. Strong percussion is necessary to bring out Williams' tone.

Second, *interrupted change of sound,* also described by Wintrich, is distinguished from the simple change, in that it occurs in different positions of the body. When the patient is in an upright position it may be present; while, if in the recumbent position, it cannot be detected, or the converse may be true. The change in position changes the relation of the bronchus to the cavity, on account of which the varying tympanitic sound is produced.

Third, *Gerhardt's change of sound.* This change depends upon the alteration of the level of the fluid when the patient assumes the upright or dorsal position. It is not necessary that the cavity communicate with the large bronchus. It is a certain symptom of a cavity, but is rare. The sound changes in pitch and in the degree of tympany. It may be absolutely dull over the lower part of the cavity when the upright position is assumed, because the fluids gravitate to this portion and come in contact with the chest wall.

Fourth, *Friedreich's respiratory change of sound.* The pitch of the sound becomes higher at the end of a deep inspiration. It depends upon increased tension of the chest wall and lung tissue as well as the wall of the cavity during the act of inspiration. It may be the cause of confusion, which is obviated by percussing at the same stage of the breathing each time, or percussing only on superficial breathing.

Fifth, Seitz has called attention to a form of breathing named *metamorphosing.* Inspiration begins harshly bronchial, then becomes faintly bronchial, the latter sound being heard also in expiration. It is said to be a sure sign of cavity.

Mensuration. By mensuration the results secured by palpation are confirmed and more accurately attained. The size of the chest is secured and its degree of expansion ascertained. If the method is resorted to from day to day it can be graphically recorded by tracing sections on paper, and delicate changes therefore definitely ascertained. The circumference of the chest is measured by means of the ordinary tape measure or by metal tapes joined together by a hinge. The latter can be made to fit accurately the circumference of the chest, and are essential in order to transfer the section to paper. The middle of the hinge is held firmly over the spinous process of the vertebra, while the two limbs are carried around the chest, moulded to all inequalities, and crossed in front, one above the other; a mark is made on each where it crosses the middle line. The measurement should be taken at about the level of the nipple, and care should be taken to have the level uniform in front and behind. The outline secured by this method need not be disturbed, as by flexion on the hinges we are enabled to remove it intact. The tapes are carefully transferred to a sheet of paper on which imaginary diameters have been marked. After fixing the corresponding points of the tapes on the lines of the respective diameters, the outline can then be traced.

Woillez's cyrtometer is a chain with links which is used to ascertain the exact circumference. The *diameter* of the thorax is secured by means of caliper compasses. The antero-posterior diameter should be taken on a level with the nipple and at the insertion of the second rib behind; the transverse diameter at the highest points of the axillæ. The length of the chest may be ascertained by measuring in the mid-clavicular line from the clavicle to the border of the ribs. It is important to remember that the right side of the chest measures a little more than the left in people who are right-handed.

The respiratory capacity is estimated by measurement of the circumference of the chest. This is secured by taking the measurement at the end of complete expiration and then at the end of complete inspiration. In health the difference between the two should be from five to ten centimetres (two to four inches). If the expansion is less than two inches it is considered deficient by insurance companies, and the risk is not regarded as first-class. The expansion is less in women. In taking the measurement the observer must be particular to keep the terminal portion of a tape measure fixed in the median line of the structure. The other portion is to be held in the hand, so as to move with inspiration and expiration. Always mark in advance the anterior mesial line and note the exact level at which measurements are made when they are taken daily.

Spirometry. By means of the spirometer Mr. John Hutchinson has been able to estimate the quantity of air taken in with each inspiration and discharged with expiration. By it the respiratory or vital capacity is estimated. The data ascertained are not of much diagnostic significance, although if measurements are made from day to day we may be able to estimate the extent of recovery from disease of the lung which was incapacitated. We can also estimate the degree of interference with breathing by disease below the diaphragm. By means of Walden-

burg's pneumotometer the respiratory pressure of air on inspiration and expiration is determined. Expiratory pressure is diminished in emphysema, and the degree of diminution may furnish a clue to the severity of the disease or the degree of improvement. It is to be remembered that it is always greater than expiratory pressure in health. It is natural to find that inspiratory pressure is lessened in stenosis of the air-passages in phthisis and in pleural effusions, although it is not of diagnostic significance.

Combination of Physical Signs. In order to determine the physical condition of the lung it is necessary to draw conclusions from the results derived by all the methods of physical examination. It is the exception that any one sign is pathognomonic of a physical condition. If the student will glance over the abnormal physical conditions which may take place in the lung he will find that they may be divided, first, into physical changes in the lung proper, and, second, into physical changes in the pleura. With regard to the lung, it will be further noted that the changes are due to an increased amount of air or to a diminution of the amount of air.

Increased amount of air may be general, unilateral, or local, and be indicated by a combination of physical signs which are usually unerring. On inspection (*a*) enlargement, general, unilateral, or local; (*b*) increased action in general emphysema, although with diminished respiratory excursion; when unilateral or local, increased action and increased expansion (compensatory emphysema). On palpation, inspection confirmed, and vocal fremitus diminished when the increased amount of air is general, slightly increased when it is unilateral or local. On percussion, in each instance exaggerated resonance or tympany. On auscultation, when general (emphysema), feeble respiratory murmur, with prolonged expiration; when unilateral or local, exaggerated respiratory murmur. The difference in the physical signs of increased amount of air are not due to the difference in quantity, but to the associate physical condition and the force of the movement of the air. The diminished expansion and feeble respiratory murmur in emphysema are due to the inability to exhale the air because of the diminished elasticity of the lung, while the occluded bronchioles from bronchitis lessen the fremitus. In cavities—local increase of air—the physical condition of the tissue which surrounds them modifies the physical signs.

Decrease in the Amount of Air. The diminution in the amount of air from change in the physical condition of the lung is due to consolidation or to collapse of the lung. The latter occurs when the bronchus is obstructed, the former in congestion, pneumonia, gangrene, abscess, forms of tuberculosis and hemorrhagic infarct. The physical signs are the same under all circumstances, except in collapse: expansion lessened, fremitus increased, dulness, bronchial breathing. The signs vary with the degree of consolidation as follows: Slight increase to greatly increased fremitus, impaired resonance to complete dulness, bronchovesicular to bronchial breathing. In tuberculosis there may be flattening of chest wall, but otherwise the signs are the same. The presence of new sounds depends upon the amount of secretion or fluid, as is the case when there is increase of air in the part.

Broadly speaking, therefore, in affections of the lung proper, the two conditions just mentioned must be differentiated—air increased, air diminished. We do not refer to bronchitis, because no physical change takes place in the lung, and the signs depend upon the amount of fluid in the tubes.

The Pleura. If satisfied that the physical condition is not due to change in the lung structure, the state of the pleura must be investigated. Here, too, the physical condition may be due to an excessive accumulation of air or to an accumulation of solid material. In effusion there is enlargement of the affected side, there is diminished movement, and also diminution of fremitus and of resonance. When air is present, however, there is tympany; when fluid, there is dulness on percussion.

The problem may be looked at from another side, however. 1. The percussion note at once indicates that there is an increased amount of air. Is this in the pleura or the lung? If in the pleura it can only be unilateral, and is recognized by the diminution of movement and of fremitus, as against increased movement and fremitus when due to unilateral increase of air in the lung proper (compensatory emphysema). 2. The percussion note, on the other hand, shows dulness or the absence of air. Is this in the pleura or in the lung? A distinction between consolidation and pleural effusion must be made. In consolidation there is increased fremitus, increased vocal resonance, bronchial breathing, and dulness on percussion. (See Fig. 44.) There may or may not be contraction. In pleurisy with effusion, absent movement, absent fremitus and resonance, dulness on percussion, feeble, distant, or absent breath-sounds. (See Fig. 45.) The distinction of the two physical conditions seems easy, and yet the physical signs may not be sufficiently definite to warrant a positive conclusion. There are cases in practice in which it is almost impossible to determine which of the two conditions is present. It has been stated previously that bronchial breathing may be present in pleural effusions. On the other hand, in certain cases of consolidation it may be absent and the vocal fremitus and resonance also absent. Apart from reliance on the associate general and local symptoms, we must look to two methods for corroborative proof of the presence of either condition. First, exploratory puncture; and, second, the involvement of organs, or change of the anatomical relations of parts. The former has been spoken of. The latter includes displacement of the heart to the right or the left, depending upon the seat of the effusion; dislocation of the liver, and, in cases of left pleural effusion, obliteration of the half-moon space (Traube's line).

Sputum. By this term is generally understood all the products of secretion of the mucous membrane of the respiratory tract, and other substances that may be brought up through the respiratory tract. The characters of sputa in disease vary with the part affected as well as with the pathological nature of the disease. It is always well to examine each specimen both *macroscopically* and *microscopically.*

Method of Collection. Sputum that is to be examined should be collected in perfectly clean vessels, containing no fluid, preferably in glass

or white earthenware spittoons, and care should be exercised against the entrance of extraneous substances, as tobacco or particles of food from the mouth, or from outside sources, or from the stomach through vomiting. Tobacco, prunes, and bread-crusts are at times mistaken for blood. It is also necessary to see that the matter sent for examination is derived from the lungs, and is not simply the oral and faucial accumulations. If practicable, the mouth and pharynx should be first rinsed with a warm alkaline solution. The true sputum is coughed up.

We usually require, in the examination, one or two glass dishes or plates, a large and a small piece of window glass, mounted needles, and forceps; and for microscopic work, in addition to these, a good microscope and accessories, and certain staining fluids.

In describing sputum we note the quantity in twenty-four hours, its color, odor, specific gravity, its composition and consistency, whether mucous, purulent, muco-purulent, frothy, watery, bloody, tenacious or viscid, and whether it is made up of separate layers or is homogeneous.

The *quantity* in twenty-four hours varies from a few c.c. to even 1000 c.c., as in a discharging empyema.

The *color* changes with the composition and the nature of the disease; thus in *acute bronchitis* and œdema of the lung it is white; in purulent sputa, no matter what the cause, it is yellow or greenish yellow; in pneumonia "rusty"; in abscess of the liver with amœba characteristics, brownish-red or like "anchovy sauce."

The *odor* is by no means characteristic in most cases. That of bronchiectasis, gangrene, and putrid bronchitis is particularly *heavy* and fœtid, a characteristic which renders its origin almost unmistakable.

The *reaction* is always alkaline.

The *specific gravity* may vary from 1.0043 (mucous sputum) to 1.0375 (serous). (Von Jaksch.)

Varieties of Sputum. *Mucous Sputum*, on account of the mucin, is usually glairy, clear, and tough. It is seen in acute bronchitis in the early stage and œdema of the lung. There is in health a small amount of mucus expectorated, which in cities and smoky towns is apt to contain black pigment particles, due to inhaled soot.

Purulent Sputum is composed almost entirely of pus. Typical purulent sputum is that from an empyema discharging through a bronchus. It may also occur in bronchiectasis, chronic bronchitis, abscess of the lung, of the liver, or more rarely of the mediastinum, discharging through a bronchus; or it may be the discharge of a tubercular vomica. The special condition can usually be determined by microscopical examination and the accompanying symptoms and signs.

Muco-purulent Sputum. It is most common to have mucus and pus mixed together in varying proportions, and then it is termed muco-purulent. Such sputa may be found in the same conditions as purulent sputa. When flat, coin-shaped masses are formed, sinking to the bottom if the vessel contains water, as in phthisis and chronic bronchitis, it is known as "nummular" sputum; or it may be more spherical, and is then called globular. At times the sputa may be seen to separate into three distinct layers, the upper frothy, muco-purulent, greenish-yellow

or dirty green, sometimes lumpy, sometimes composed of shreds; the middle thin and watery, with shreds from the upper layer; and the bottom layer, apparently made up of pus and débris, opaque and without air-bubbles. It points to *gangrene* of the lung in most instances, but may also occur in *bronchiectases.*

Watery or serous sputum is the result of œdema of the lung.

Bloody Sputum—Hœmoptysis. As blood in sputum is always of importance, the entrance of substances as mentioned above, which simulate it in appearance should be guarded against. It may be seen in greatly varying quantities and have many different sources, and have slight or grave significance. It may come from the gums, nose, pharynx or larynx, and in all cases such sources should be examined. Again, there may be cases where bleeding from the stomach (hæmatemesis) simulates hemorrhage from the lungs, and still more often people speak of vomiting blood that really has come from the lungs. Usually that from the lungs is much more frothy and bright red, while that from the stomach is darker and acid, and may contain particles of food. Diagnosis is more difficult when some blood from the lungs is first swallowed, then vomited. Usually there is a distinct history of preceding cough, and for some time afterward small amounts of blood continue to be expectorated. (See Lungs: hemorrhage.)

Small amounts of blood streaking the mucous sputum or appearing in small clots often come from the throat or nose or upper air-passages, but may come from the lungs. Muco-purulent sputum streaked with blood frequently is indicative of phthisis. In pneumonia the rusty sputa are the result of an admixture of mucus and blood, and usually contain small air-bubbles. When the blood-coloring matter is changed there may be a yellowish or greenish tinge. In certain cases of chronic pneumonia, where the blood remains longer in the lung tissue, the expectoration has a darker color. Where there is slight leakage from an aneurism the same may be found. Pneumonia accompanied by expectoration of large amounts of blood is often indicative of a tuberculous origin. Blood may be mixed with the greenish expectoration of gangrene. According to Finlayson this is especially true in children. In chronic valvular disease of the heart, frothy mucus containing more or less blood is commonly seen, and likewise in oozing from aneurism. "Currant jelly" sputa are more or less characteristic of malignant growths of the lungs, while the expectoration from a liver abscess with amœbæ is reddish-brown in color, from the mixture of blood, pus, and bile elements, and is not unlike "anchovy sauce." We may have hemorrhage from the lungs as a part of a general hemorrhagic tendency, as in purpura and hemorrhagic smallpox; and in so-called "vicarious menstruation" there may be hæmoptysis. But a patient presenting such symptoms should be examined with the greatest care to exclude actual pulmonary complication. When great quantities of blood are expectorated we suspect tuberculosis of the lung, aneurism, or cardiac valvular disease.

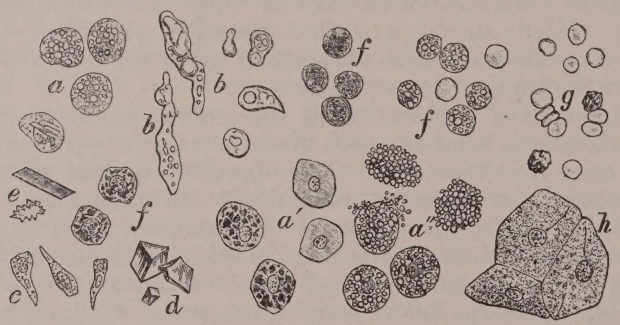
The unaided eye may distinguish other foreign substances, such as fibrinous casts of the bronchi or trachea and spirals; but full consideration of them will be given farther on.

Microscopic Examination of the Sputum. *White blood-corpuscles* are present in all sputa, but in varying numbers and size. They are most abundant in purulent sputa. Often they contain fat drops and pigment particles.

Red blood-corpuscles are to be found in the majority of sputa. They may be so few as not to give a red color. The source is often high up in the respiratory tract. When in considerable number the sputum is more or less tinged, and in hæmoptysis it is almost wholly made up of red cells. Usually each cell is well preserved, but they may be recognized as pale bodies or as rings, the pigment remaining in the sputum as pigment particles or as crystals of hæmatoidin, as in pneumonia.

Epithelium. Two general varieties are found in the sputum—squamous and cylindrical. The former comes from the mucous membrane of the mouth, the tongue, tonsils, true vocal cords, and perhaps from the salivary and small bronchial glands. It has no clinical importance.

Fig. 52.



Epithelium, leucocytes and crystals of the sputum. (Eyepiece III., obj. 8, A. Reichert.)

a, *a'*, *a''*, Alveolar epithelium. *b*, Myelin forms. *c*, Ciliated epithelium. *d*, Crystals of calcium carbonate. *e*, Hæmatoidin crystals and masses. *f*, *f'*, *f''*, White blood-corpuscles. *g*, Red blood-corpuscles. *h*, Squamous epithelium. (Von Jaksch.)

Cylindrical cells in sputum are rarely perfect. It is uncommon to find the cilia intact, and still more so in motion, while the body of the cells is likely to be changed. They are found in inflammations of the trachea and bronchi, or the posterior nasal fossa—a locality where, it must be remembered, ciliated epithelium exists.

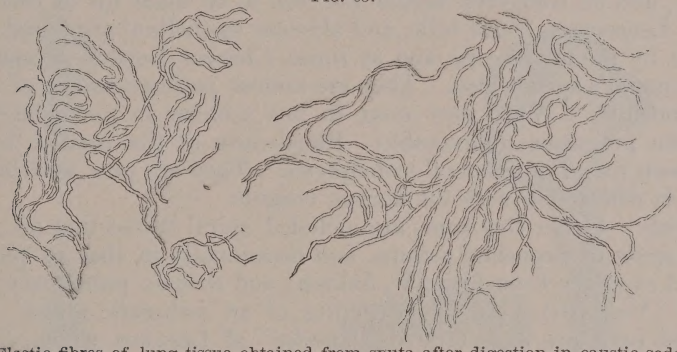
"*Alveolar*" *epithelium*, so called, when found in the sputum, is more important than the above, as different observers consider its presence to have more or less clinical significance. The cells are elliptic or round, somewhat larger than white corpuscles, with a single nucleus, which is indistinct without the addition of acetic acid. The protoplasm is granular, and contains within it particles of iron-dust, carbon, or blood-coloring matter, and often fat drops. They may also have undergone complete fatty degeneration, and they have been considered the source of myelin drops in the sputum.

It is to be found in the sputum of chronic bronchitis, acute and chronic pneumonia, and tuberculosis of the lung.

Detection. A small bit of sputum is placed on a microscopic slide and a cover-slip applied. Examine with varying powers, and again after acetic acid is added stain the cells with an aqueous solution of methylene-blue.

Elastic fibres. As the presence of elastic fibres in sputa is of much import, denoting destruction of the lung tissue, bronchi, or the larynx or bloodvessels, their presence from food remaining in the mouth must be especially guarded against. They may be mistaken for fat crystals. They are found as single threads in bundles, or showing an alveolar arrangement. They are to be recognized by the double contour and curling ends, and at times by the alveolar arrangement. They may be due to tuberculosis, abscess of the lung, bronchiectasis, gangrene of the lung, pneumonia (Von Jaksch) and rarely to destructive diseases of the larynx. In a very great majority of cases they are due to tuberculosis. It is uncommon to find them in gangrene, probably because, as Traube first suggested, they are destroyed by a ferment.

FIG. 53.



Elastic fibres of lung tissue obtained from sputa after digestion in caustic soda. (Drawn by DR. JOHN WILSON.)

Elastic tissue from the alveoli often shows the diagnostic alveolar arrangement; the fibres that form a bronchus are branched; those from an eroded artery appear in the form of a network, or the fibres are bound together.

Detection. The method employed by Osler, modified from Sir Andrew Clark's, is the best. A small amount of the thick, purulent portions of sputum is pressed out into a thin layer between two pieces of plain window-glass, 15 x 15 cm. and 10 x 10 cm. The particles of elastic tissue appear on a black background as grayish-yellow spots, and can be examined *in situ* under a low power. Or the upper piece of glass is slid off till the piece of tissue is uncovered, when it is picked out and examined on a microscopic slide, first with a low power, as the one or one-half inch objective, and then with a higher power. At first there will be some difficulty in distinguishing with the naked eye between elastic fibres and particles of bread or milk globules, or collections of epithelium and débris, but with practice such mistakes are rarely made, and the microscope always reveals the difference. This method is much easier of accomplishment and quite as satisfactory in

results as the one generally employed—boiling an equal quantity of sputum and solution of caustic potash (8 to 10 per cent.) for a short time, and allowing it to stand for twenty-four hours in a conical glass. The elastic tissue remains intact and is found in the sediment.

Connective tissue and *cartilage*, in fragmentary bits, are rare constituents of sputum. The former may occur with abscess or gangrene of the lung, and the latter when there is ulceration of the larynx.

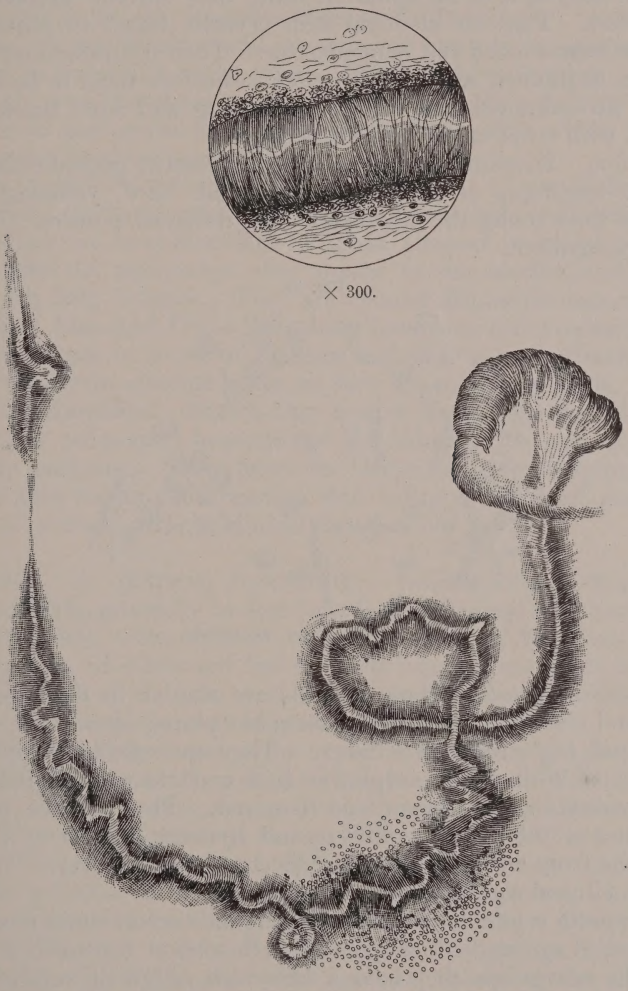
Fibrinous Coagula. These striking tree-like bodies are found in the sputa of plastic bronchitis, and at times in that of pneumonia, phthisis, and in diphtheria and croup where there has been an extension into the bronchi. They are usually mixed with mucus and are rolled up into a mass. Their peculiar form is best seen when they are washed and unravelled in water. They are then seen to be a complete mould of a small bronchus with its ramifications. The size varies greatly. They may be many centimetres long. In fibrinous bronchitis the size and shape of the moulds in different attacks may be exactly similar, as if they came from the same bronchus. They are grayish-white in color, hollow, and on transverse section are seen to be made up of cast upon cast. Leucocytes, blood cells, and alveolar epithelium are found in the meshes by the microscope, and at times Charcot–Leyden crystals and Curschmann's spirals also. They are almost pathognomonic of fibrinous bronchitis. When they occur in any number in pneumonia they make the prognosis unfavorable. Blood-casts of the smaller bronchi have been found in cases of hæmoptysis. They are rare, and have no apparent connection with the fibrous coagula.

Spirals. Under this name are included spiral bodies that are found in the sputa of bronchial asthma, and occasionally in that of pneumonia and capillary bronchitis (V. Jaksch), and chronic pulmonary tuberculosis (Vierordt). At the beginning of an asthmatic attack, tough rounded balls are expectorated—"*perles*" of Laennec, which, if freed of the mucus surrounding them and spread out on glass with a dark background, may be seen by the naked eye to have a twisted spiral form. With the aid of the microscope they are found to be made up of spirally arranged mucin in a more or less tight twist, with many cells from the alveoli and bronchi. In some of these spirals a shining central thread runs through the entire length of the spiral like a core, remarkable for its clearness and its high refractive index. The fine fibres composing the spiral may be closely arranged or not. Epithelium and Charcot-Leyden crystals may be found lying among the coils. The main constituent of the spirals is mucin, and Osler has suggested that the central thread is made up of transformed mucin. On the other hand Von Jaksch believes it to be chemically distinct from the mucin spiral. Vierordt considers it either made of tightly twisted central fibres or to be an optical image of a core cavity. They are probably the result of an acute bronchiolitis. Why they should assume this remarkable form is still an open question. It has been suggested (Osler) that the ciliated epithelium of the bronchi may have a rotatory action, and their action combined with the spasm of the bronchioles tends to the spiral formation.

That there is a connection between the spirals and Charcot-Leyden

crystals seems very probable, as the latter are absent from the sputum at the beginning of an attack of bronchial asthma, but if a portion of such sputum is allowed to stand for twenty-four to forty-eight hours, so that evaporation does not take place, crystals will be found. As has been said, the crystals are often found among the spirals, and this when

FIG. 54.



× 300.

Spirals from bronchial tubes. × 80. (After LEYDEN.)

they are seen nowhere else. Later on the spirals disappear, but crystals derived from them (?) continue to be expectorated. (See Fig. 54.)

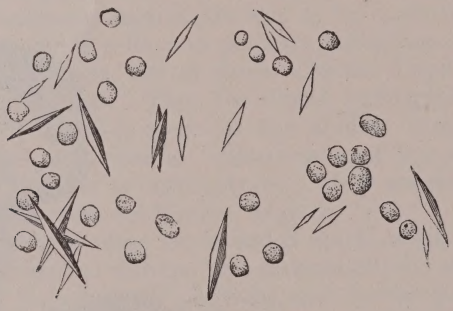
The method of examining for spirals is as given above.

CRYSTALS. Charcot-Leyden, cholesterin, hæmatoidin, fatty, tyrosin, oxalate of lime and triple phosphate crystals are to be found in sputa under various conditions.

Charcot-Leyden crystals are octahedral, sharply pointed, colorless, or slightly bluish, soluble in warm water, alkalies, and acetic and mineral acids. The practised unaided eye may recognize these as small yellowish bodies, not unlike grains of sand; under the microscope they are unmistakable. Their size varies greatly. They occur most abundantly during (invariably) and after an attack of bronchial asthma; they have also been seen in the sputa of acute and chronic bronchitis and tuberculosis. They are identical with crystals found in semen, fæces, and in leukæmic blood and bone-marrow. Their connection with spirals has been mentioned above. Schreiner considers them to be the phosphate of an unknown base, which Ladenburg and Abel think may be identical with ethyl eninim. (Von Jaksch.)

Detection. Examine the sputum of an asthmatic patient a day or two after the beginning of an attack for round, hard, yellowish bodies, and place these under the microscope with different powers. They are readily recognized.

FIG. 55.



Charcot's crystals. (SCHEUBE.)

Cholesterin crystals. These crystals are similar to those of cholesterin found elsewhere, being thin rhombic plates, often with irregular corners and high refractive index. They are soluble in ether; and when treated with dilute sulphuric acid and tincture of iodine they become violet, blue, or green, and then red. They may be present in the sputum of tuberculosis, abscess and hydatid abscess of the lung, and in pus from an abscess of another organ, as the liver. They have but little clinical significance.

Hæmatoidin crystals. Hæmatoidin crystals are at times recognizable by the naked eye as distinct spots of yellowish or brownish red color. Under the microscope they have a brownish yellow or ruby-red color, and are either in the form of small rhomboid prisms or of fine needles, single or arranged in bunches of various shapes, or as free pigment particles without crystalline form; smaller particles may be contained within a leucocyte. Their presence indicates that blood has remained in the respiratory tract for some time before being expectorated, or that an abscess has discharged into a bronchus. They occur in phthisis, following hemorrhage; in thoracic aneurism when blood is oozing into the lung; in gangrene; in abscesses discharging through a bronchus. Von Jaksch

states that when the crystals are contained in cells there has been a preceding hemorrhage, but that when there is considerable free hæmatoidin one infers that an abscess of a neighboring organ has discharged into the lung.

Fatty crystals. Crystals of margaric acid occur as long, thin needles, gently curved or bent at one end much like a fish-hook, and either singly or in bundles. They are found in unhealthy pus—as in gangrene, putrid bronchitis, bronchiectasis, and tuberculosis ; in the plugs formed in inflamed tonsils ; and in purulent sputum in general which is allowed to stand in a warm place. They dissolve in ether and boiling alcohol ; and this characteristic, together with the regularity of their curve, should distinguish them from elastic fibres, with which they are sometimes confused by beginners.

Tyrosin crystals have been found in the sputum of putrid bronchitis and empyema discharging into the lung, and usually in conjunction with leucin. They are most abundant in sputa allowed to stand for some time. Under the microscope they appear as fine needles, and can be mistaken for fatty crystals. They are without diagnostic importance.

Oxalate of Lime and Triple Phosphates have been noted occasionally in sputa ; the former in a case of diabetes and also in an asthmatic ; the latter occur only in alkaline sputa, as they are soluble in acids.

Corpora Amylacea. Starch-like bodies have been found in the sputum after pulmonary hemorrhage has taken place, and in that of pulmonary gangrene. They have the shape of starch corpuscles, and sometimes give the amyloid reaction with iodine or iodide of potassium. They are at present without clinical significance.

Parasites. A. Animal Parasites. Echinococcus cysts are to be found in sputum, generally broken into fragments, and only very rarely in a perfect whole, when there is rupture of a cyst of the liver or lung into a bronchus. Scolices and free hooklets from the same may be recognized, and pieces of the cyst wall will be known by their remarkable formation. Their presence is of great clinical value.

Infusoria have been found in the expectoration from gangrene of the lungs. They belong to the monad and cercomonad varieties.

Distoma hæmatobium eggs may occur in sputa when the lung tissue is broken down by its presence, the eggs being thrown off in the sputum.

Amœba Dysenteriæ (*Amœba Coli*). Of far more interest and importance is the presence of this parasite in the expectoration. A full description of the amœba will be given in the chapter on the Fæces. They are the same in every respect when found in the sputum, except that they are often slightly larger. The sputum containing the amœba is partly diffluent, tenacious, frothy, bright red in color at first, due to the presence of blood, and later brick or brownish red, sometimes bile-stained. Small yellowish white cheese-like particles are seen. Upon exposure to the air the sputum becomes thin, syrupy, and oily, and it then looks much like anchovy sauce. The sputa are alkaline, and of a faintly sweetish odor, never putrid. Later on they become more purulent, somewhat nummular, reddish yellow, and contain less blood. If there is a favorable termination they become more fluid and frothy, with less blood and pus, and on standing show the three layers. The

quantity varies from 25 c.c. to 500 c.c. in twenty-four hours. Under the microscope will be found, besides the amœba, red blood-corpuscles, leucocytes, alveolar and oval epithelium, and bodies looking like degenerated liver cells without a nucleus; and occasionally elastic fibres, hæmatoidin, leucin, tyrosin, and Charcot-Leyden crystals and bacteria are seen. The cheesy particles are made up of amorphous granular matter and oil globules. Amœbæ are constantly present in varying numbers, usually not so many as in the stool, but somewhat larger. The number varies from day to day, and diminishes with the disappearance of the cough and expectoration. The sputa should be examined as soon after their discharge as possible, and in the interim should be kept at a temperature of 30° to 35° C. If examined on a warm stage active movements of the amœbæ will be kept up much longer.

They should be examined under various powers: $\frac{1}{2}$, $\frac{1}{5}$ or $\frac{1}{7}$, and $\frac{1}{12}$ inch objectives. Of these the $\frac{1}{5}$ or $\frac{1}{7}$ inch will be found most suitable for following the movements. They measure from 10 μ to 20 μ. They will be readily recognized by their size, formation and movements. (See Fæces for further description.) That they have important clinical value is true, as cases have been reported in which the observer diagnosticated hepatic or hepato-pulmonary abscess secondary to amœbic dysentery, by the peculiar anchovy-sauce expectoration and subsequent detection of the amœbæ.

B. Vegetable Parasites. *Fungi—Non-Pathogenic: Moulds. Oïdium albicans* may be a constituent of the sputum when the bronchi are invaded by it, but usually it is from the saliva. Certain other moulds have lately been considered to cause disease of the lungs by multiplication, but nothing very definite has resulted from the experiments thus far made.

Yeast fungi. Von Jaksch reports having seen scattered yeast-cells in the pus from a phthisical cavity. Otherwise we have no knowledge of yeast being found in sputa.

Fission Fungi. Leptothrix. Leptothrix occurs alone in the sputum or in the bronchial plugs in putrid bronchitis, along with fatty acid and hæmatoidin crystals. It is probably derived from the mouth, having thence entered the air-passages, or it is taken up from the mouth by the expectoration. It is recognized by its staining blue with iodine and potassium iodide.

Sarcinæ pulmonalis. Sarcinæ may be seen in sputa. They are larger than sarcinæ ventriculi, with which they have no connection, nor have they pathological significance when present in sputa.

Non-pathogenic bacilli and cocci may occur in all sputa, but are without significance. They are more numerous in fœtid sputa. They stain with methylene-blue and other simple dyes.

Pathogenic Fungi. Tubercle Bacillus. The organism which is the cause of tuberculosis is a rod, straight or slightly curved, without motion, varying in length from 2 μ to 5 μ (about $\frac{1}{4}$ to $\frac{1}{2}$ the diameter of a red corpuscle). It usually has a beaded appearance when stained, due to the spores present, which do not take up the stain that affects the rod as a whole, and which often slightly bulge beyond the edge. The bacillus of tuberculosis cannot be recognized in the sputum unless

stained, and in the staining it shows a peculiarity which belongs to only one other organism—the bacillus of leprosy. As under ordinary conditions this latter bacillus is not met with, this peculiarity in staining is diagnostic of tubercle bacilli.

Preparation of Sputum and Method of Staining Tubercle Bacilli. A small amount of the purulent portion of the sputum is spread in a thin and uniform layer on a perfectly clear cover-glass by means of forceps, needles, or the "Oese,"[1] which must previously be held a moment in the flame of a Bunsen burner or spirit lamp; or by pressing a small amount of sputum between two cover-glasses, then sliding them apart. It is then dried in the air, or more quickly by holding the cover-glass with forceps some distance above the flame of a burner or lamp. Finally, it is to be passed three or four times through the flame, and so "fixed." The edge of the cover-glass, with sputum side up, is then grasped with forceps and covered with the staining solution, care being taken to prevent the fluid from extending to the under surface, and held in or just above the flame, until the solution boils for a second or two or a bubble rises. When the excess of the solution is washed off in water, the slip is treated with the decolorizing agent until the color is almost or wholly removed. It is again washed in water to remove the excess of the decolorizer, and mounted for examination, or given a contrast stain; the latter is preferable. If fuchsin has been used to stain the tubercle bacilli, methylene-blue is a good contrast stain; while if gentian-violet was selected, Bismarck-brown is better in contrast. These contrast stains are made as needed by dissolving enough of the dye in a few c.c. of water to make the solution as seen through a test-tube of 14 cm. diameter only transparent, and then filtering; or a concentrated watery solution may be made for stock just as the concentrated alcoholic solution of fuchsin and gentian-violet were made, diluting a small quantity of this when needed with enough distilled water to make it just transparent in a similar test-tube. To apply the contrast stain, place a few drops of the same on the cover-glass that has been prepared as above—stained, decolorized and washed—allow it to remain for thirty or forty seconds, wash off in water, and mount for examination on a glass slip, in water, oil of cloves, or Canada balsam. A drop of water will serve perfectly well for examining when the preparation is not to be preserved. In the microscopic examination use a $\frac{1}{12}$ inch oil-immersion lens and Abbe condenser, or, at the least, a $\frac{1}{7}$ or $\frac{1}{8}$ inch objective. If gentian-violet has been used the tubercle bacilli appear as dark blue rods, with all other bodies brown, if Bismarck-brown is used for contrast stain; while with fuchsin staining for the tubercle bacilli, and methylene-blue as a contrast, the former will be found as red rods in a blue field (background). (See Plate I., Fig. 1, A.)

The above rapid method of staining takes much less time than the method usually described, and gives most satisfactory results. The steps in the old method are the same as given above, except that instead of placing the staining solution on the smeared and dried cover-glass, and

[1] This most useful instrument is made by fusing a piece of glass rod 10 to 15 cm. long and inserting into the fused end a piece of platinum wire about 5 cm. long. The free end of the wire remains straight, or, better still, is bent into a loop.

holding it in or above the flame until the solution boils, the cover-glass is floated in a cold solution in a watch-glass, sputum side down, for twenty-four hours, or in a hot solution for six to eight minutes, or until moisture appears on the upper surface of the cover-glass. The remaining steps are similar.

Tubercle bacilli do not stain with the simpler dyes, but when stained by solutions of dyes made more penetrating by the addition of aniline oil, carbolic acid, or like substances, *they retain the color when subjected to decolorizing agents.* In this they differ from all other organisms, except, as stated, the bacillus of leprosy.

A number of methods have been devised for the detection of this bacillus by means of its peculiar action toward stains. The most satisfactory are those known as the Koch-Ehrlich, Ziehl-Neelsen, Gabbett, and Gibbes. These methods differ chiefly in the solutions used. Slightly modified from the original in execution, they are as follows:

A. Koch-Ehrlich method.

Solutions Used.

I. Concentrated alcoholic solution of fuchsin or gentian-violet.
II. Saturated solution of aniline oil in water.
III Thirty per cent. solution of nitric acid in water (decolorizing solution).

I. Place in a clear bottle fuchsin or gentian-violet in substance to one-fourth its capacity, and fill with alcohol (95 per cent.); shake well and cork and allow to stand for twenty-four hours. If all of the dye has been dissolved add more and shake, and stand for another twenty-four hours, and so on until some of the dye remains permanently undissolved at the bottom of the bottle. This solution remains good until used.

II. To about 100 c.c. of distilled water in a flask or other suitable vessel, add aniline oil, drop by drop, shaking the flask continuously, until the solution is opaque, or drops of the oil float on the surface, then filter through moist filter paper until the filtrate is perfectly clear. This solution must be made fresh as needed.

III. Mix a few c.c. of nitric acid and water in about the above proportion, never stronger, each time bacilli are to be stained.

The Koch-Ehrlich solution is made by adding 11 c.c. of the fuchsin or gentian solution (No. I.), and 10 c.c. of absolute alcohol to 100 c.c. of the clear aniline filtrate (No. II.). It should not be used after it is a week old.

B. Ziehl-Neelsen method.

Solutions Used.

I. Carbolic-fuchsin solution:

Distilled water	100 c.c.
Carbolic acid (crystalline)	5 grammes.
Alcohol	10 c.c.
Fuchsin in substance	1 gramme.

This solution can also be prepared by adding saturated alcoholic solution of fuchsin (see above) to a 5 per cent. watery solution of carbolic acid until a metallic lustre is seen on the surface of the fluid. This solution does not decompose so easily as those made with aniline oil. It should not, however, be over ten to fourteen days old.

II. Decolorizing solution of nitric acid, and
III. Contrast stain of methylene-blue, as above.

The preparation and staining are exactly the same as in method *A*. The tubercle bacilli are stained red, the other bodies blue.

C. Gabbett's method.

Solutions Used.

I. Carbolic fuchsin solution (as in *B.*).
II. Methylene-blue solution:

Methylene-blue[1]	2 grammes.
Sulphuric acid	25 c.c.
Distilled water	75 c.c.

This solution is liable to decompose if old.

Preparation of Slips and Staining. The cover-glass is prepared and stained with the carbolic-fuchsin solution and washed in water as in *A*. Then (instead of decolorizing with nitric acid or adding in contrast stain) the slip is washed for one-half to two minutes in the methylene-blue solution, until a faint blue replaces the red tinge in the (slip) sputum; the excess of the solution is washed off with water, and the slip is mounted and examined as above. The tubercle bacilli are stained red and the other bodies blue.

The writer has found that this method can be rapidly applied, and that it has given good results; he recommends it highly.

D. Gibbes' method.

Solutions Used.

I. *a.*	Fuchsin	3 grammes.
	Methylene-blue	1 gramme.
	Mix thoroughly in a mortar.	
b.	Aniline oil	5 c.c.
	Alcohol	20 c.c.

Dissolve and add *b* to *a* slowly, stirring vigorously until *a* is evidently dissolved, then add 20 c.c. of distilled water and keep in a stoppered bottle, ready for use.

Prepare slip and stain with this solution, as with the others, up to the point of decolorizing. Then wash with alcohol until dye ceases to come away. Mount and examine as above. Tubercle bacilli will be stained dark red—the other objects dark blue.

When the bacilli are few in number, Biedert proposes that the following preliminary steps be taken: About 4 c.c. of sputum are mixed with 8 c.c. of water and 1 c.c. of solution of caustic soda, and boiled a few minutes, when about 15 c.c. of water are added and the whole again boiled until a homogeneous fluid is formed. This is allowed to stand in a conical glass for twenty-four to forty-eight hours, when the sediment is stained by the Ziehl-Neelsen or Gabbett method. Or the homogeneous fluid can be put at once in a centrifugal machine, and the resulting sediment stained.

Sputa have been hardened and sections made and stained for tubercle bacilli, but the method is not of special value.

It is well to remember that in the absence of a proper decolorizing agent, hot water applied for some minutes has been shown to decolorize very satisfactorily.

Importance. The greatest importance attaches to the presence or continuance of tubercle bacilli in sputa. It indicates tuberculosis of the lungs or larynx; in the vast majority of cases, of the former.

[1] An alcoholic solution of methyl-blue should first be made, and then added drop by drop, with constant stirring, to the sulphuric acid and water.

They are often to be found in the sputum when physical signs are still absent or are indefinite. The number varies so greatly in different cases, and in the same case at different times, that in recent cases it is impossible to judge of the extent of the disease by the number present in a given preparation.[1]

The absence of bacilli from sputa has no true value unless negative results are obtained after many trials and careful examination by an experienced observer using good stains. Hence, too great care in each and every step cannot be taken.

Biological Properties. The tubercle bacillus is difficult to cultivate, as it grows readily only in exact conditions found within the body. The best medium is blood-serum. The cheesy mass from the sputum or the tubercular nodule from a tissue is placed on the surface of the serum and rubbed carefully over it. It is best to make twenty or thirty such inoculations. The tubes must then be sealed to prevent evaporation and drying, and exposed for twelve days to a temperature of 37.5° C. When a pure culture is obtained, further cultivations may be made on agar-agar to which 6 per cent. of glycerin has been added.

The pure cultures appear as dry masses on the surface of the medium, either as flat scales or clumps of mealy-looking granules. They are of a dirty drab or brownish gray color. (See Plate II., Fig. 6.) The bacillus is parasitic, aërobic, non-motile (facultative anaërobic).

Micrococcus Lanceolatus. Pneumococcus. Diplococcus Pneumoniæ. The causative factor of croupous pneumonia is in its typical form an oval coccus, with one end smaller and more tapering than the other. It may, however, be regularly oval, or spherical. It is 1 to 1.5 μ in length, and one-half or one-third greater than its width. Forms occur in which the width is only one-half or one-fifth its length. It is thus really a bacillus, and is called such by many observers. Two cocci are usually found together, end to end, hence the term diplococcus; and often two or three such pairs are arranged together to form a chain. These chains are at times not distinguishable from some varieties of streptococci, and there may be a close connection between them. The lanceolate cocci have a capsule, a fact which aids in the diagnosis of this bacterium more than the pair arrangement or lance-shape. (See Plate I., Fig. 1, B.)

Pneumococci are stained in cover-glass preparations with the ordinary aniline dyes, as given above. The capsule may be stained and differentiated in the same way, but it more often requires a special method. Welch recommends the following: Spread and dried cover-glass preparations are treated first with glacial acetic acid, which is allowed to drain off, and is replaced (without washing in water) with aniline oil-gentian-violet solution. (See under Tubercle Bacilli.) The staining solution is repeatedly added to the surface of the cover-glass until all of the acid is displaced. The specimen is now washed in a weak salt solution (about 2 per cent.), and examined in the same, not in balsam.

[1] A Method for the Examination of the Actual Number of Tubercle Bacilli in Tuberculous Sputum. By George H. F. Nuttall, M.D., Ph.D., The Johns Hopkins Hospital Bulletin, May, 1891. The method is of pathological but not of diagnostic interest.

The capsule and coccus can then be differentiated. Degenerative and involution forms are constantly met with. There will be variations in size and shape, and the capsule may contain only remains of a coccus, or be entirely empty.

The micrococcus lanceolatus is not motile, and never forms spores. It is facultative anaërobic. It grows in various alkaline media. Favorable temperature 35° to 37°, death-point 57° C. The growth is very rapid in liquid media, rendering the fluid cloudy in six to twelve hours. After about forty-eight hours the multiplication stops and the micro-organisms settle, leaving the fluid clear. In gelatin stab cultures small white colonies form along the puncture. It does not liquefy the gelatin. On agar it forms very characteristic jelly-like drops.

By *inoculation* into susceptible animals a typical fibrinous pneumonia is developed. The pathogenic power attenuates rapidly in cultures, but recovers its virulence by passing through susceptible animals.

This micro-organism is found in all cases of croupous pneumonia. It is also found in health in the saliva, in empyemas due to its presence, making a favorable prognosis, and in meningitis, ulcerative endocarditis, acute abscesses, otitis media, and arthritis due to it.

Bacillus of Influenza. This micro-organism is found in purulent sputum. It was first detected by Pfeiffer. The bacilli have the form of minute rods, single or in chains of three and four, and stain well in Löffler's methylene-blue fluid and in the dilute Ziehl-Neelsen fluid. Cultivations have been made on glycerin agar. When solidified obliquely, separate colonies form, which, after twenty-four hours appear like drops of water visible only by a lens. In the blood they can be detected in cover-glass preparations after staining. After having the dried cover-glass preparation in an absolute alcohol bath for five minutes, then stain from three to six hours in eosin-methylene-blue fluid. (See formula.)

Actinomyces. When the lungs or pleura are infected by this fungus, actinomyces will be found in the sputum. The disease in these organs is rare. Macroscopically they appear as small kernels, yellowish white or greenish yellow, and having the shape of a millet-seed. Under the microscope they are recognized by the rounded club-like bodies projecting from all sides of a central unformed mass. They are seen better when not stained.

Chemistry of Sputum. As the chemical examination of the sputum does not aid us in diagnosis, it is of but little or of no value. Mucin, nuclein and serum albumin are constituents of sputa in health. Peptone is present whenever there is pus, and is specially marked in pneumonia. Volatile fatty acids, such as butyric and acetic, occur at times, markedly so in pulmonary gangrene. Glycogen has been obtained by Solomon, and a ferment resembling one of the pancreatic ferments has been detected, especially in pulmonary gangrene and putrid bronchitis. Of inorganic substances, chlorides of soda and magnesia; phosphates of soda, lime and magnesia; sulphates of soda and lime; carbonate of soda, lime and magnesia; and in a few cases phosphate of iron and silicates rarely obtained. (Von Jaksch.)

The Data Obtained by Inquiry.

The Subjective Symptoms. DYSPNŒA. Dyspnœa, in its true sense, means difficult breathing. The respirations are deeper than natural, but of normal frequency, or they may only be more frequent than they should be, or they may be both deeper and more frequent. The patient is usually conscious of suffering or of some distress in breathing. *Lung disease* without *dyspnœa*: While a common, indeed almost constant symptom of lung disease, it does not follow that because a patient has extensive disease of the lung he need suffer from difficult or hurried breathing. This arises because the demands of the system require no more air than the capacity of the lung is able to supply. The change takes place very gradually, but many persons with chronic fibroid phthisis, or with emphysema, in both of which the disease may be extensive, may not have dyspnœa, unless there are unusual demands upon their systems. The subjects are under-weight, move slowly, and otherwise show that they are deprived of an essential to active being.

VARIETY OF DYSPNŒA DEPENDING UPON CAUSE:

I. Anything which cuts off or lessens the normal amount of air required for oxygenation of the blood. A. Obstruction of the air-passages. B. Diminution of air-space from causes within and outside of the thorax. C. Interference with the action of the muscles concerned in breathing.

II. Affections which lessen the amount of blood, as obstructive heart disease. Rarely, tumors pressing upon the bloodvessels.

III. Affections in which the red blood-corpuscles are diminished—anæmia.

IV. Pulmonary embolism and thrombosis. In cases of weak heart the vessels become occluded. After labor a clot of blood may escape from a uterine sinus, be carried to the right heart, and thence to the pulmonic veins. The clot may arise from inflammation of the veins in any situation.

V. Foreign substances in the blood, as fat, occurring in parturient women three or four days after labor, after fractures, and in diabetes.

VI. Dyspnœa due to interference with the nervous mechanism of respiration. *a.* Tumor, hemorrhage, or degeneration about the respiratory centre in the medulla. *b.* Irritation of the centre by toxic agents, as in uræmia, diabetes, auto-intoxication from gastro-intestinal disorder. To this class belongs "heat dyspnœa," which occurs in all febrile conditions. The warm blood acts as a direct irritant to the respiratory centre in the medulla oblongata (Landois). This explains the dyspnœa of fever and the curious fact pointed out by Cohnheim, that the respirations in pneumonia lessen as soon as the fever disappears, notwithstanding the persistence of the physical condition, on account of which the dyspnœa might have been explained. Reflex dyspnœa (asthma, *q. v.*) belongs to this variety. The dyspnœa of hysteria is of the same class.

Anything which cuts off or lessens the normal amount of air required for oxygenation of the blood. A. Obstruction of the air pas-

sages. 1. Occlusion of the nares, unless compensated for by mouth-breathing. 2. Enlargement of the tonsils, retro-pharyngeal abscess, or any obstruction in the throat, from diphtheritic or œdematous swelling. 3. Disease of the larynx causing stenosis also causes a characteristic form of dyspnœa (see Disease of the Larynx). 4. Obstruction of the trachea or bronchus from external pressure or from a foreign body. Dyspnœa from the latter cause must be distinguished from dyspnœa the origin of which is higher up in the air-passages. Inspection of the upper cavities usually reveals the cause.

a. Tracheal Obstruction. In dyspnœa from occlusion of a bronchus or the trachea there is no increase in the movement of the larynx. There is no change in the voice, except that it may be weakened and the sonorous quality diminished. If, however, there is at the same time attendant disease of the larynx from syphilis, or paralysis of the muscles from pressure on the recurrent laryngeal nerves by the same cause which produces the tracheal stenosis, the voice will be modified. If so, on laryngoscopic examination the tumor pressing upon the larynx can be seen at times, especially if the larynx is healthy. Expert operators can secure a considerable view of the windpipe, particularly if the head is bent slightly forward and the patient is seated in the upright posture. A mirror must then be placed against the soft palate with the surface more horizontal than usual. By this means an aneurism may be seen bulging into the trachea. It must not be mistaken for pulsation of the lower end of the trachea due to transmission of the impulse of the aorta to the trachea, which has been shown to occur in healthy persons.

The dyspnœa is expiratory and is never so extreme as in laryngeal stenosis. The lower ribs are therefore not sucked in during inspiration until late in the disease. A stridor attends the dyspnœa which is heard with the stethoscope over the trachea, as well as over every part of the chest. Sometimes a point over the trachea can be determined at which the sound is heard loudest. The point may indicate the seat of a stenosis. Sometimes the sound is more marked over the larynx than over the sternum when the lower part of the trachea is obstructed. Demme has pointed out that in cases of prolonged obstruction in the lower air-passages the upper portion of the thorax may diminish in size. The dyspnœa is not only constant, but paroxysms may also take place in which the distress is very severe. These paroxysms of dyspnœa may be due to spasm of the vocal cords; but it is very likely that they are due, as Bristowe has shown, to swelling of the mucous membrane or to mucus which has accumulated at the point of obstruction and cannot be dislodged, or to spasm of the muscular tissue of the trachea itself. In addition to the subjective symptom of want of breath the patient may complain of pain or oppression behind the sternum, or possibly only of a slight soreness. Cough usually attends the dyspnœa, with expectoration of mucus. Sometimes the mucus is blood-tinged, and even streaks of blood may be expectorated after a considerable time, in cases of leaking aneurism.

If the obstruction is due to a *foreign body* the dyspnœa occurs *suddenly.*

b. Bronchial Obstruction. If a bronchus is obstructed the lung to which air passes freely becomes the seat of extensive emphysema. When obstruction takes place gradually compensatory emphysema occurs, developing slowly, not rapidly as in the former instance, the degree depending upon the amount of obstruction in the opposite bronchus. When the bronchus is obstructed the physical signs are pronounced. The vesicular murmur over the corresponding side of the chest is absent, fremitus is absent, the movement of the affected side is impaired. With these changes the percussion sound is normal. As the case advances the affected side may fall in and measure less than the opposite side. A snoring or whistling sound may be heard over the root of the lung, between the scapula and vertebræ, or moist râles may be present. The causes of tracheal and bronchial obstruction are: first, tumor of the *thyroid gland;* second, *thoracic aneurism;* third, *mediastinal tumor* from other cause than aneurism, as disease of the glands, cancerous or tubercular, or mediastinal abscess; fifth, *cancer* of the *œsophagus;* and, finally, in rare cases, a *dilated auricle.* But diseases of the walls of the trachea also cause obstruction by narrowing the calibre. *Syphilis* is the most frequent cause of such obstruction. Within the lumen the presence of a *foreign body* causes obstruction. The foreign body may remain free for a time, moving up and down as the patient coughs, and, indeed, it may be felt against the side of the trachea when the finger is placed outside the neck. Later the foreign body usually becomes fixed in the right bronchus, or one of its main divisions, because the opening of the right bronchus is more direct than that of the left. In some instances the body may be dislodged and fall into the opposite bronchus. Rarely it falls first into the left.

B. *Dyspnœa from Diminution of the Air-space in the Lungs.* All forms of pulmonary disease attended by consolidation, by compression of the lung, or occlusion of the small bronchi, are included under this subdivision. The degree of dyspnœa of course depends upon the extent of the diminution in the air-space. In *pleural effusions* from any cause the air-space is lessened and dyspnœa occurs. In bilateral effusions it is more marked than in unilateral. The severity of the dyspnœa depends somewhat upon the rapidity with which the effusion takes place. In cases of sudden effusion of air, as in *pneumothorax,* the dyspnœa is very alarming at first, but as accommodation takes place it is gradually relieved. In rapid effusion of serum it is also serious.

The characteristic form of dyspnœa due to lessened air-space is seen when obstruction of the air-tubes takes place on account of spasm. The attack comes on suddenly in the midst of quiet breathing (see Asthma). It occurs in paroxysms in asthmatic subjects. It may occur, however, on slight exertion, or it may in a measure be constant. But when the dyspnœa that is associated with asthma is constant other changes have taken place in the lungs. First, there is persistent bronchitis; second, the presence of emphysema. Indeed, in many cases it is often difficult accurately to ascertain the sequence of affections. In emphysema of the lungs dyspnœa is constant, but on exposure to cold or on account of an attack of indigestion, more severe paroxysms may occur, and asthmatic attacks, although the patient is not an asthmatic. On the other

hand, a patient may have had asthma for a long period of years, during which attacks of dyspnœa occur in paroxysms only. As time passes the paroxysms become more and more frequent, on account of which emphysema develops. With the advent of the emphysema the dyspnœa becomes more constant.

Asthma is a type of dyspnœa of nervous origin. It has just been said that it is due to spasm of the bronchial tubes. This may occur from a number of causes: (*a*) It may be of central origin, from irritation of the pneumogastric centre; (*b*) it is just possible that some disturbance of the trunk of the pneumogastric nerve will also cause asthmatic dyspnœa; but what concerns us most is (*c*) the paroxysmal dyspnœa which arises reflexly from irritation of the terminal endings of the pneumogastric nerve, or of nerves intimately associated with the pneumogastric, in the medulla. First. Disease in the upper air-passages, as polyps, or a hypertrophy of the turbinated bones, or adenoid growths, are the most frequent source of paroxysmal dyspnœa. Not only in permanent disease of this character do we have such dyspnœa, but temporary irritants applied to the nares likewise produce it. Various odors, the irritation of micro-organisms, or of pollen, or emanations from vegetable life, provoke attacks of nasal congestion and reflex dyspnœa. The irritation is propagated through the ethmoidal and posterior nasal branches of the nerve, the Vidian and naso-palatine nerves to the septum, and the anterior palatine to the middle and lower turbinates. Second. Irritation in the fauces and larynx is not so likely to cause dyspnœa, yet there is no doubt that the presence of a constant irritant in these situations tends to provoke, or keep in a state of excitability, the respiratory tract, so that asthma is more likely to persist. Third. To this class of cases belongs the irritation of the terminal branches of the pneumogastric nerve in the stomach. Peptic asthma, or the asthma of indigestion, may owe its origin to these causes. Often the irritation is central, due to the irritating influence of an abnormal product of indigestion upon the respiratory centres in the medulla. Fourth. For the same reason we have asthma due to other poisonous substances circulating in the blood, as the poison of uræmia. The dyspnœa due to this condition usually occurs in paroxysms, but may become constant. Sometimes it is the first intimation of the presence of renal disease. The dyspnœa of diabetic coma may occur from the same cause. The nature of both of them is recognized more particularly by their associate symptoms. The condition of the urine, the odor of the breath and the exhalations, the presence of hypertrophy of the heart and of an accentuated second sound, point to a uræmic origin. The history and symptoms of diabetes, the odor of acetone on the breath, the presence of sugar in the urine, the absence of organic disease, point to diabetes. The dyspnœa of uræmia cannot be distinguished from other forms of dyspnœa, except by the exclusion of cardiac and lung disease. The latter is difficult often, because uræmia so frequently develops after the hypertrophied heart has failed, so that the physical signs of dilatation may be sufficient to explain the dyspnœa. The dyspnœa of diabetic coma, known as "air-hunger," is characterized by slow and deep respirations. The Cheyne-Stokes respiration has for its source the same cause, namely, irritation

in the medulla, as in other forms of nervous dyspnœa. It must not be forgotten that the dyspnœa of uræmia may present the Cheyne-Stokes phenomenon.

Diminution of Air-space from Extra-pulmonary Causes. Anything which crowds upon the thorax, interfering with pulmonary expansion, causes dyspnœa. In affections below the diaphragm, this is notably the case. Hence in enlargement of the various organs of the abdomen, as the liver, spleen, kidneys, pancreas (cystic disease), and uterus, dyspnœa always occurs. In accumulations of gas (flatulency), or of fluid (ascites), the diaphragm is pressed upward and encroaches on the thoracic capacity. In abdominal tumor, as of the ovary, the omentum, and of the organs above mentioned, dyspnœa is a distressing feature.

C. *Interference with the Action of the Muscles.* Practically any derangement of the action of the respiratory muscles diminishes the air-space, as expansion of the lungs is interfered with. Nevertheless the cause of the dyspnœa is extra-pulmonary. It is due to weakness or paralysis of the muscles concerned in breathing, or to inhibition of their action on account of pain, or to interference with their action on account of obesity, myxœdema, or œdema, or on account of actual disease, as in trichinosis or myositis.

1. Phrenic dyspnœa is a peculiar form due to *paresis* of the *phrenic nerve* and consequently to interference with the action of the *diaphragm.* It may not be observed as long as the patient is at rest. Upon slight exertion the effort distresses him and causes an increase in frequency of the respirations. After a few steps a sense of suffocation ensues, or upon ascending an elevation the patient must stop frequently to take a breath.

Other physiological processes are affected in phrenic dyspnœa. In the act of sighing the patient feels as though the abdominal organs were drawn up into the chest. Any straining effort, as at defæcation, is embarrassed. The voice is weak, and there is difficulty in coughing and sneezing, because a full inspiration cannot be taken. A slight attack of bronchitis may be very serious on this account. On inspection during inspiration, instead of the natural expansion of the ribs and chest, the epigastrium and the hypochondriac regions are drawn in. During expiration they are pushed forward. The thoracic movements are reversed. The abnormality may be detected on palpation with both hands below the cartilages of the ribs, even better than by inspection. Unilateral paralysis of the diaphragm causes drawing in of the corresponding hypochondriac region.

In progressive muscular atrophy, in general lead-poisoning, and in multiple neuritis from other causes, paralysis of the diaphragm may take place. It is said to occur in hysteria, and Walshe states that he has seen it after diphtheria. In fatty degeneration of the diaphragm, on account of inflammation extending from the peritoneum to the pleura, the same phenomenon has been seen. It may occur in trichinosis.

Paralysis must be distinguished from inaction of the diaphragm. When the drawing in during the act of inspiration of one or both hypochondriac regions occurs it is diagnostic of the occur-

rence of inaction rather than paralysis; whereas paralysis of other muscles, with a distinct cause for paralysis, is found with the latter condition.

The dyspnœa that occurs from paralysis of other respiratory muscles can be recognized on careful inspection and palpation. The atrophied groups of muscles are readily observed. Electricity may aid in the diagnosis.

2. Pain inhibits muscular action. The source of the pain may be in the pleura, the muscles, or the intercostal nerves. Frequently it is below the diaphragm, as in peritonitis, hepatitis, etc., interfering with the action of that muscle. The dyspnœa that occurs from pain, as pleuritis, or inflammation of the chest wall, is recognized by the posture which is taken in order to relieve the affected side, by local tenderness, and by the physical signs of pleurisy or of pleurodynia.

Clinical Varieties. We observe whether dyspnœa is (*a*) modified by exertion, (*b*) attended by alteration in the respiratory rhythm, (*c*) is constant or paroxysmal.

(*a*) *Influenced by Exertion.* 1. *Shortness of breath* may be apparent on *exertion* only, as in cases of simple debility, or of interference with respiratory action on account of obesity. It is the form of shortness of breath seen in anæmia and in moderate cardiac debility. It may not be observed by the patient unless he walks hurriedly or ascends a flight of stairs. 2. *Shortness of breath independent of exertion* is of more serious import, and is due to a number of causes. It is the shortness of breath that is seen in severe cardiac and pulmonary disease. To the latter belong asthma and emphysema, bronchial obstructions, pulmonary consolidation and compressions (by effusions).

(*b*) *The Rate of Respiration.* Dyspnœa varies clinically, depending upon the frequency of the respiration. In its most extreme form it is known as *orthopnœa*, when the upright posture of the trunk is assumed. (See Posture.)

1. Dyspnœa *with respiration slow or normal.* *a.* Dyspnœa may be characterized by deep inspirations, the frequency of respiration being less than normal. This is one of the forms of dyspnœa seen in diabetic coma—"breathlessness without dyspnœa." It is most characteristic, and associated with nausea, vomiting, and coma, while the breath and urine smell of acetone. *b.* The breathing may be slow and stertorous. Such breathing is likewise associated with coma, but the coma is of central origin, due chiefly to apoplexy or tumor.

2. *Irregular respiration.* Alternately slower and shallower breathing, and then quicker as well as deeper, is seen in the peculiar form of breathing known as the Cheyne-Stokes respiration. It includes a period of apnœa, and at the same time alterations in the size of the pupils. (See Uræmia and Diseases of the Brain.) 3. *Respirations increased.* The respirations may be hurried and create distress in simple nervousness alone, and hurried respiration is quite common in cases of hysteria. Often in the latter instance the frequent breathing is not attended by distress to the patient. The respirations are panting in character, and are half the normal pulse rate or even as much as the pulse. The term panting is applied to such respiration at times. The same character

of breathing is seen in exophthalmic goitre. The rate of respiration is increased in all forms of dyspnœa upon exertion (see above), and all forms due to heart or lung disease. It may be observed that slow respirations with dyspnœa are usually central or toxic. Toward the end of life the respirations, even though hurried before, become slower from carbon dioxide intoxication.

(*c*) Dyspnœa may be further divided clinically into *constant* and *paroxysmal* dyspnœa. Constant dyspnœa implies a persistence of the cause. Paroxysmal dyspnœa does not include the form that is increased by exertion—a form which in one sense may be paroxysmal. It is seen in its most typical form in asthma. It is often of cardiac origin, or may be due to central or reflex causes. It occurs usually at night. Constant dyspnœa is frequently subject to aggravations paroxysmal in occurrence. Asthma is the type of true paroxysmal dyspnœa.

Diagnosis. While dyspnœa is usually easy of recognition, it must not be forgotten that attacks of acute indigestion with thoracic symptoms of oppression may simulate the oppression of dyspnœa. It is temporary, however, and not associated with increased rapidity of the respiration. Dyspnœa is recognized by increase in rapidity of chest movement, with increased action of all the muscles of respiration, both the essential and the auxiliary muscles. At the same time the expression is pronounced. The alæ nasi move, the eyes and countenance are indicative of more or less agony, the pupils are dilated. As the dyspnœa continues cyanosis develops, and frequently a cold sweat breaks out. This may be limited to the forehead and face and to the extremities, or may become general. The hands and feet become cold. Stupor sets in, carpopedal spasm or general convulsions follow, the respirations become slower, and death takes place in coma or from heart failure (asystole).

The dyspnœa of emphysema is characteristic. The difficulty is seen to be due to the inability to empty the chest of air (expiratory dyspnœa). The inspiration is short and quick; the expiration is prolonged, and all the auxiliary muscles are called upon to complete the act. The powerful abdominal muscles are seen to contract vigorously, and thus aid in pressing up the diaphragm. The quadratus lumborum and serratus posticus superior et inferior draw down the ribs. The scaleni are strongly contracted, the serratus magnus, latissimus dorsi, and the pectorales all aid in elevating the ribs. Knowledge of the processes involved in forced expiration render the diagnosis comparatively easy. The contraction of the broad abdominal muscles confirms the diagnosis.

Cough in Pulmonary Affections. (See Larynx.) Coughing is a reflex act. A deep inspiration is taken, followed by closure of the glottis, succeeded immediately by a sudden expiratory effort during which the glottis is opened, causing a loud sound with the forcible passage of air outward, along with any substances in the air-vessels. The pulmonic irritation, on account of which the act takes place, usually starts in the respiratory mucous membrane. The cough is then used to expel accumulations of mucus or pus, or foreign substance. It occurs in all forms of bronchitis and in the lung affections generally in which bronchitis is associated. The cough of phthisis, if not laryngeal, is due to a local-

ized bronchial catarrh. Nodules outside of the bronchi, situated in the lung substance, do not provoke the act of coughing, as we see in the calcareous and fibrous nodules of healed tuberculosis. The irritation is not limited to the mucous membrane of the bronchial tubes but occurs in the mucous membrane of any portion of the respiratory tract. A foreign body of any kind in the bronchus sets up cough. It is notably present in pharyngeal and laryngeal diseases. The cough of the latter is of peculiar character, which renders it easily distinguished from cough due to other causes. Cough may also occur from causes outside of the air-passages. It may be of centric origin. Kohts has found by experiment that irritation of the floor of the fourth ventricle above the centre for respiration excites a cough. It is possibly on account of this centric origin that we may explain the cough of hysteria and the short barking cough which arises in hysterical or nervous states when the patient is afflicted with the idea that he is about to have hydrophobia. Irritation of nerves which are in anatomical relation with the pneumogastric also excites cough. The most characteristic cough of this form is that due to the presence of a foreign body in the meatus of the ear, or to disease of that organ. It is sometimes difficult to examine the external auditory meatus, because coughing is excited. The afferent nerve which receives the irritation is the auriculo-temporal branch of the fifth nerve, according to Dr. Fox, and not the minute auricular twig of the vagus.

Tooth Cough. The same authority points to the occurrence of cough from the irritation of the stump of a tooth, and refers to cough in infants during the first dentition.

Stomach Cough. The popular opinion that cough is very frequently due to the stomach is not substantiated by the experiments of Kohts. We, nevertheless, have a cough very frequently with patients who are suffering from mild gastric catarrh, the treatment of which relieves the cough. This is in all probability due to the fact that with the gastritis there is a secondary pharyngitis, and as the former is relieved the latter, which causes the cough, disappears entirely.

It will be seen, therefore, that when investigating the cause of a cough in diseases in which this symptom is prominent it is necessary not only to make examination of the respiratory tract throughout its course, but also to examine the condition of the ear and the teeth, and to bear in mind its possible centric origin.

Clinical Characteristics. The cough may be dry or moist. 1. A *dry cough* occurs when there is an irremovable source of irritation. (See dry cough of laryngeal disease.) It is seen in the first stage of *bronchitis.* It occurs in the earlier stages of *phthisis.* As a short, hacking, suppressed cough it occurs in *pleurisy* in the first stage. In the second stage it is superficial, as if the sound waves were checked. It is characteristic and most familiar, although described with difficulty. It is particularly the type of cough due to irritation outside of the respiratory tract. The ear cough and tooth cough partake of this character. In cases of emphysema the cough may be dry and unproductive for a long time, and only be relieved after a small pellet of tough mucus is discharged. In the same category belongs the nervous cough which occurs from bad habit, the cough of hysteria, and the cough

of a peculiar barking character that occurs at puberty, which Sir Andrew Clark has described.

2. The *moist* cough is attended by expectoration of a mucous, muco-purulent, purulent, or bloody character, which is comparatively easily removed. Dry and moist or loose cough may be either constant or paroxysmal, or both. The moist cough may occur in paroxysms only, each paroxysm being relieved by the removal of the irritation, the subsequent paroxysm not taking place until the irritating secretion has reaccumulated. In cases of *bronchitis* of the second stage paroxysms of cough may occur every few hours, or the cough may take place once in the twenty-four hours, usually in the morning on arising. The accumulated secretions of the night are disposed of, and then the patient remains free from annoyance. In some circumstances the cough is almost constant. The irritation is constantly present. A large amount of secretion is rapidly poured out, keeping up a constant cough. This is seen in *bronchorrhœa* and bronchial dilatation and in the later stages of *tuberculosis*. In these affections the moist cough may occur three or four times in twenty-four hours, during which time an enormous amount of sputum is thrown off. The cavity is thereby emptied, the accumulation of matter in which excites coughing only after a certain level is reached. In this affection the cough is further characterized by aggravation on change of position. In *pertussis* the character of the cough is of special diagnostic significance. In this affection the cough occurs in paroxysms. The expiratory efforts are frequent and rapid, followed by a noisy, prolonged inspiration, during which the characteristic whoop is created. At the same time the appearance of the countenance is marked. The face is cyanosed, the eyes stare, the appearance of distress is most striking. The labored efforts at coughing frequently terminate in an attack of retching or vomiting.

It must not be forgotten that the presence of an irritant does not always excite cough. Thus when the reflexes are obtunded, as in typhoid fever, in disease of the brain, or in the last stages of any disease, the presence of mucus will not excite cough, and yet it is known to be in the trachea, on account of the rattling which takes place. In cases of phthisis, sudden checking of the cough and expectoration, on account of weakness, is of bad prognosis and denotes approaching death. It is also a bad sign in pneumonia.

The Sound. The character of the sound of the cough is usually modified by the condition of the larynx, for which reference must be made to the section on laryngeal diseases.

The *diagnostic* significance of cough is estimated by the character; by the sound; whether constant or paroxysmal; by the frequency of the paroxysm; by its development at particular times or under particular circumstances, as on rising in the morning, or change to a cold atmosphere, or speaking, or upon movement, as in phthisis. By the sound, laryngeal and bronchial coughs are distinguished. Constant cough implies a persistence of the cause, which is strictly pulmonary, as in pleurisy, phthisis, bronchitis, and consolidations generally; paroxysmal, a recurrence of cause when pulmonary, or a reflex or central cause. Paroxysmal coughs occur in cavities, either of the lung or of

the pleura opening into the lung. Cough is excited whenever the cavity fills with secretion. The paroxysm may occur daily or several times a day. Paroxysmal cough occurs in bronchitis after a certain amount of secretion accumulates. It is the cough of irritation outside of the lung, excited by reflex influences. The association with retching and vomiting is of some diagnostic significance. It is not only seen in whooping-cough, but is of frequent occurrence in phthisis. The value of cough in diagnosis is enhanced by knowledge as to the duration of the cough and by the character of the expectoration. (See Sputum.)

Hemorrhage. Hemorrhage of the lungs occurs from disease or from rupture of adjacent bloodvessels into the air-passages. It is not alone a symptom of lung disease. A hemorrhage may be small in amount and continue over a considerable period of time, or it may be characterized by a sudden profuse discharge, which at once terminates the life of the patient.

Cause. A. Affections of the lungs. 1. Anything which causes *congestion of the lungs* will lead to hemorrhage. In this instance the amount of blood is small. It may be limited to streaking of the expectoration, or a few mouthfuls may be discharged. In (*a*) *organic heart disease* this form of hemorrhage is seen. It is also a characteristic feature of the first stage of (*b*) *croupous pneumonia.* The rusty-colored sputum is due to the rupture of the capillaries. In (*c*) *hemorrhagic infarcts* hemorrhage occurs, and, with the sudden formation of a consolidated area in the lung, is diagnostic. In (*d*) *phthisis* it also occurs (see below).

2. *Tuberculosis.* In tuberculosis hemorrhage may occur either (1) as the first symptom of the disease, on account of collateral congestion around infiltrated areas, or (2) later, on account of ulceration of an artery when excavation of the lung has taken place. In the early stages the hemorrhage is usually profuse, but not fatal. It may occur repeatedly during a series of weeks, excited no doubt by the violent nonproductive cough which attends the earlier stages of this disease. In the later stages, when the vessels are ulcerated, the patient may have repeated hemorrhages, varying from a few ounces to half a pint or a pint. They may occur daily, or be repeated at intervals of a week or more for a long period of time. After the hemorrhages that occur at long intervals the patient experiences much relief. Indeed, the dyspnœa, cough, and chest oppression subside in a remarkable degree, and the occurrence of another hemorrhage is often predicted by gradual recurrence of these symptoms. Death does not usually ensue on account of the large hemorrhage from phthisical ulceration, and yet it may possibly take place. The writer has seen four instances of hemorrhage into a large cavity, in three with external hemorrhage also, which caused death instantaneously. 3. Hemorrhage recurring frequently is significant of *cancer* of the lungs, in the absence of other causes. 4. It is of common occurrence in *plastic bronchitis*, when large bronchial casts are expelled. 5. In *gangrene* of the lung it frequently occurs, often causing death. The odor and sputum indicate the true nature of the primary lesion. 6. Hemorrhage with the expectoration of calca-

reous masses occurs and recurs frequently in patients with healed or *quiescent tubercle.*

B. Disease outside of the respiratory tract. (1) Aneurismal disease of the bloodvessels which have intimate relation with the trachea and bronchus frequently causes ulceration into these tubes with the occurrence of hemorrhage. The hemorrhage is usually profuse and often induces sudden death. Sometimes the profuse hemorrhage may be preceded for days by small hemorrhages. The physical signs of aneurism are sufficient to explain the cause of the hemorrhage. The bleeding can sometimes be seen in the trachea when an aneurism of the innominate artery or the aorta presses upon that tube. (2) In diseases of the heart it does not take place generally until the later stages of the disease, and is associated with secondary congestion of the lungs. It may, however, be an early symptom in mitral stenosis. The hemorrhages may amount only to staining of the sputum, or several times during the day an ounce or more of blood may be expectorated.

C. Affections of the blood or bloodvessels with hemorrhages in other portions of the body. Thus, it may occur in hæmophilia, in the forms of purpura, in scurvy, and in anæmia. It occurs in jaundice with hemorrhages in other situations.

D. Gouty endarteritis. In the aged in both sexes, hemorrhages take place independently of disease of the heart or of the parenchyma of the lungs. Sir Andrew Clark and others have spoken of these hemorrhages and attributed them to gouty changes in the vessels as well as to degenerations of lung tissue, on account of which the rupture took place.

E. Without known cause. In certain instances pulmonary hemorrhages occur in which it is quite difficult to find any cause for the discharge. It is quite common to see hemorrhage occur in females: sometimes at the menopause, in other cases during menstruation, or, again, perhaps vicariously when menstruation does not occur. A number of cases that have been under the writer's observation have had this tendency for years without the development of pulmonary disease, and, apparently, without much influence on the general health. Indeed, it may be said that hemorrhage from the lungs in women, other things being equal, is not of grave significance.

The Symptoms. The only symptom may be the presence of blood in the expectoration, or the discharge of a small amount of blood with slight cough. In either instance, unless the patient's mental condition is rendered obtuse by disease, the hemorrhage is alarming to him. Much perturbation is created, and with other nervous phenomena, palpitation of the heart may take place. Apart from the nervousness excited by the sight of the blood, small hemorrhages, and even hemorrhages of moderate amount, do not cause any other symptoms. The symptoms of a large hemorrhage depend upon the amount of blood that is lost. They may amount to faintness and giddiness only, or with them pallor may ensue. If more pronounced, syncope may take place; extreme pallor develops; the pulse becomes rapid, small, and feeble; the extremities are cold, and the face bathed in perspiration. If the syncope is recovered from, the patient is extremely restless, sighing

and breathing hurriedly. There may be some nausea. Moderate delirium and mild febrile symptoms often follow the restlessness. If the hemorrhages do not recur and the patient's fears are calmed, the color will gradually return and the heart's action become stronger and slower. These symptoms occur whether the hemorrhage is due to disease of the lungs or to aneurism rupturing into the bronchus. If the hemorrhage is large they differ somewhat in the two conditions. If a large aneurism ruptures, the blood rapidly wells up into the throat and pours out through the nostrils and mouth with great rapidity. With such hemorrhage the fatal end may come in a few minutes. In pulmonary hemorrhages the discharge is not so profuse, and is attended by the act of coughing. With each cough blood is raised to the amount of a full mouthful at a time. The blood discharged from the lungs is bright in color, very frothy, being mixed with air. There are no clots in the discharged fluid. The blood from an aneurism is also bright red, but is not frothy, unless the discharge is very slow, and becomes mingled with air in the vessels. In rare cases of pulmonary hemorrhage an abundant stream of blood pours out, which is dark in color, free from clots, and without mixture of air (large cavity).

Diagnosis. Hemorrhage from the lungs must be distinguished from hemorrhage from the upper air-passages and from the stomach and œsophagus. Thus a discharge of blood from the mouth may occur from cracks in the pharynx, or varicose veins. It is not abundant and the hemorrhage is mingled with mucus, which is streaked with the blood. Hemorrhage from the gums may be taken for pulmonary hemorrhage, but if there is no stomatitis or inflammation of the gums from scorbutus or ptyalism, the source of the blood can easily be traced. In stomatitis its color is somewhat different. It is thin, fluid blood, often offensive, of cherry-juice color. Hemorrhage from the lungs is distinguished from hemorrhage from the stomach by the difference in the method in which it is discharged, and difference in the character of the blood. In hemorrhage from the stomach the blood is vomited. It is mixed with particles of food or other gastric contents. It is dark in color, often of the appearance of coffee-grounds; it is not mixed with air, and hence is not frothy. The rapid hemorrhage from ulceration of an aneurism into the œsophagus, or rupture of varicose veins at the lower end of the œsophagus, cannot be distinguished from the hemorrhage that occurs when the aneurism ruptures into a bronchus. The recognition is dependent upon the physical signs and the previous history of the patient's illness.

Pain. Pain is rarely a symptom of disease of the lungs unless the pleura is involved. In a case of bronchitis there may be some soreness and oppression behind the sternum, but otherwise pain is absent. In pleurisy, pain occurs before the exudation. The pain is sharp and lancinating, and so severe as to impede respiration and cause the cough to be short and catchy. It is usually seated at the base of the chest in the lateral or anterior region. It occurs when the patient attempts to take a full breath. Before the inspiratory excursion is half completed it is checked involuntarily on account of the pain. The patient's hand is placed upon the affected part and he involuntarily leans to that side.

The pain of pleurisy may be increased by local pressure, but general pressure, as from the whole hand, a broad bandage, or a large strap of adhesive plaster always gives relief. In the pleurisy that attends phthisis the pain is quite common. It is of the same character as the pain of acute plastic pleurisy, but varies in situation and in degree. The pain occurs in paroxysms. It follows a slight exposure to cold, undue exertion, or fatigue. It may continue for twenty-four hours, to remain away until a repetition of the cause institutes it again. It must be distinguished from the myalgia of phthisis due to cough and exposure. In myalgia, the muscles and fasciæ at the bony attachments are very tender.

The pain of pleurisy must be distinguished from pleurodynia, from intercostal neuralgia, and from the pain due to disease of the ribs. In *pleurodynia* the muscles are sensitive if pressed between the fingers or palpated. An enlarged area is affected, but physical signs of pleurisy or pneumonia cannot be elicited. Cough is absent, and so usually is fever. It is associated with pain in other muscular or fibrous structures. There may be a history of exposure to cold and dampness preceding it. Usually there is a history of lithæmia or frequent myalgia in the patient. *Intercostal neuralgia* is sometimes difficult to distinguish. The pain is sharp, localized, and may modify the movements of the chest. General pressure relieves it, local pressure at the points where the terminal filaments of the nerve come to the surface may be detected. The so-called Valleix's tender points are, however, not always present in cases of intercostal neuralgia. The patient is usually anæmic, often the subject of uterine or other exhausting disease, and may suffer from neuralgia in other situations. Cough and physical signs are absent. *Fracture of the ribs* or caries of the rib may be recognized by the local tenderness, and by the signs of these conditions. Localized pleurisy may attend both, however—indicated by more severe pain on cough or full breathing. Caries or fracture is determined by pressure upon the diseased rib, and by the crepitus of fracture. An empyema that is about to point will cause pain in some area of the chest. The pain usually is seated at the points of election for the discharge of the empyema, and is soon followed by swelling, with heat and redness of the skin, and the occurrence of œdema.

More or less constant pain at the apices, undoubtedly independent of affections of the muscles, is a suspicious sign of tuberculous disease in that situation. It may be aggravated by pressure.

Special Diagnosis.

Diseases of the Bronchi. Diseases of the bronchi are distinguished from other diseases of the lungs chiefly by the difference in the physical signs. Except in capillary bronchitis, the general and subjective symptoms are not so severe as in other affections.

We are aided in the recognition of bronchial affections, first, by the fact that they are bilateral; second, that the bases are usually affected; third, that there is diminution of fremitus determined by palpation;

fourth, that there is absence of dulness on percussion; fifth, that râles are more pronounced in proportion to other physical signs, and more general than in other lung affections.

Bronchitis.

Bronchitis is an inflammation of the mucous membrane of the bronchial tubes. It may be acute or chronic, may involve any part of the bronchial tree, the large, the middle-sized, or the most minute branches, and may be primary or occur secondarily to some general disease or to disease of the heart or kidneys.

1. Acute Bronchitis occurs most frequently by extension of the catarrhal inflammation from the nose and throat; but in some persons it develops so suddenly that it appears to be primary in the tubes.

When the *larger* or *middle-sized tubes* are involved the patient complains of soreness or rawness underneath the sternum, especially at its upper part. There is frequently a feeling of tickling in the throat, and a sense of weight or oppression on the chest. Chest pain is due to myalgia or the strain upon the muscles from coughing. The *cough* is at first hard and dry, and often produces pain of a tearing character in the muscles of the chest or abdomen. The cough is apt to be worse when the patient first lies down, and again on rising, especially after a night's rest. *Fever* is usually slight and of short duration. The respirations are accelerated, but not markedly, and dyspnœa does not exist. The *expectoration* is at first a white, frothy, viscid mucus, subsequently becoming more abundant and muco-purulent.

Physical Signs. In uncomplicated cases there are no changes in the physical structure of the lungs. On examination of the chest, the percussion note is found to be clear; the respiratory murmur more roughened and harsh than normal, but not broncho-vesicular or bronchial; accompanying breathing there are heard sibilant and sonorous râles, and, in the later stages, some large and medium-sized mucous râles. The râles vary in position from time to time, and especially after coughing. Vocal resonance and fremitus are unaltered. A fremitus may be produced by sonorous râles.

The cough and expectoration usually last for some time after fever has subsided. The duration of the disease is from a few days to several weeks. It is never fatal except in the very old and very young, or in those who are much debilitated.

The *diagnosis* of acute bronchitis is easily made by noting the fact that the disease runs an acute course, marked by fever, cough, and expectoration; and that the physical signs are negative except as to roughening of the respiratory murmur and the existence of bronchial râles heard on both sides of the chest.

From *croupous pneumonia* and *local tuberculosis* of the lungs it is distinguished by the absence of dulness on percussion, bronchial breathing, and increase of vocal resonance and fremitus; by the absence, in other words, of the ordinary signs of consolidation. From pneumonia it is further to be distinguished by the milder character of the subjective symptoms and by the fact that in bronchitis the physical signs

are almost always bilateral, in pneumonia generally unilateral. From *tuberculosis* it is further to be distinguished by the slow progress of the latter, which involves the apices preferably, whereas bronchitis is more marked at the bases; and by the occurrence sooner or later of hectic fever and emaciation, which are absent in bronchitis. Doubt will exist usually only at first; the progress of the case will in time make everything clear. Systematic examination of the *sputum* is an important diagnostic aid, and will lead to the differentiation of many cases of bronchitis from tuberculosis and from pneumonia. In infants and children especially, bronchitis is at times so rebellious to treatment that tuberculosis is suspected.

In *broncho-pneumonia* (catarrhal pneumonia) there is a diffuse bronchitis associated with small areas of pneumonic consolidation. It is to be distinguished by the graver general symptoms and by detecting small areas presenting dulness on percussion and bronchial breathing, associated with the physical signs of bronchitis already described.

Acute miliary tuberculosis of the lungs is very liable to be mistaken for bronchitis, because dulness, if present, amounts to nothing more than tympanitic dulness, because the signs are diffused through both lungs, and the respiratory murmur is fainter than normal but only slightly roughened. Close inspection of the patient will, however, make it evident that he is more ill than could be accounted for by bronchitis alone. The fever is higher, the respirations more frequent, pallor, with a dusky or faintly cyanotic hue intermingled, is common, perspirations are more pronounced. A primary focus for the process may be discovered or a source of infection ascertained.

Acute bronchitis may be mistaken for *spasmodic laryngitis* (croup). It is to be distinguished by the less amount of spasm and by the presence of fever in addition to the physical signs. In bronchitis the breathing is rarely so stridulous as in laryngeal spasm.

Whooping-cough cannot be distinguished positively from bronchitis before the characteristic whoop appears; but it may be suspected when the child has been exposed to contagion, and when the coryza and redness of the fauces persist in spite of treatment.

In the diagnosis of bronchitis it is often more difficult to determine the primary cause of it than to distinguish it from other pulmonary affections. The former is most important. It needs to be borne in mind that bronchitis is a frequent accompaniment of many febrile diseases, such as typhoid fever, measles, and whooping-cough; of diseases of the heart and kidneys, and of septic diseases and blood disorders. The primary will not be likely to be mistaken for the secondary disorder if one is upon his guard and insists upon finding a cause for each case that presents itself.

Measles can be diagnosticated from the first usually by the coryza, but especially by the red spots upon the anterior half-arches of the soft palate, which appear usually several days before the eruption.

Bronchitis is a common and important early symptom of *typhoid fever*. The latter disease may be suspected when the fever, prostration, and headache are greater, and especially if these symptoms coexist with a loose condition of the bowels, chilliness, and occasional nose-bleed.

2. Capillary Bronchitis, or Suffocative Catarrh, is bronchitis of the smaller tubes. It occurs most frequently as the result of extension of the catarrhal process from the larger tubes, but sometimes seems to attack the smaller tubes from the beginning, or coincidently with the larger tubes. Infants, young children, and the aged are more liable to it. It begins with a succession of chills or chilliness, followed by high fever. The temperature may rise to 104°. The skin is hot, the face flushed. The head and neck and the upper portion of the trunk may be covered with perspiration. The pulse is rapid and soon increases to great frequency.

The aspect of the patient from the first shows that the illness is graver than that of ordinary bronchitis. The face expresses anxiety, and in children the alæ nasi play in respiration, which is both considerably accelerated and difficult (*dyspnœa*). The respirations may run as high as 60 or 80 to the minute, the pulse not being correspondingly frequent Dyspnœa is more or less constant, but becomes urgent in paroxysms, and the patient may need to be propped up in bed in order to breathe (*orthopnœa*). It is *expiratory:* inspiration may be free and easy, or it may be difficult, but expiration is always difficult and prolonged. In children the pause in the act of breathing takes place at the end of inspiration, instead of expiration.

Cough is more frequent and violent than in ordinary bronchitis, and the expectoration viscid and difficult to raise. As the disease progresses dyspnœa becomes more intense, and signs of deficient aëration of the blood make their appearance (*cyanosis*). The lips and finger-nails become bluish, and the extremities cool and clammy. If the patient is unable to expel the tenacious secretions from his bronchial tubes the further progress of the case is that of rapidly developing cyanosis; the breathing continues frequent, but is shallow and more labored. Children are liable to have convulsions, followed by coma and death, while old persons sink into coma without preceding convulsions.

On the other hand, if the case is favorable, the patient's strength is maintained, and he is able to cough hard and expectorate, consciousness is unclouded, and cyanosis does not become marked.

The physical signs are those of bronchitis of the larger and smaller tubes; sibilant and sonorous râles, if present at first, give way to fine subcrepitant and crepitant râles, which speedily become moist and very abundant. As in ordinary bronchitis, the bases of the lungs posteriorly are the parts most involved. The percussion note remains clear over both lungs, but there is apt to be increased resistance. The fremitus may be lessened in some areas, increased in others. If an area of dulness appears it may be due to pneumonia or collapse of the lung; if the former, there is usually an access of fever.

The *sputum* contains mucus, pus, occasionally blood-cells, granular matter, and sometimes fibrinous casts of the tubes.

3. Chronic Bronchitis occurs most frequently in middle or later life. Its special features are its long duration, without fever, and with comparatively little impairment of the general health. Cough is not constant; there are periods when it is entirely absent; the disease then returns, perhaps with increased severity, and lingers indefinitely.

Chronic bronchitis consists in its milder form in what is often called "winter cough." It attacks especially persons past middle life, who have emphysema. It appears at the onset of cold weather, and lasts until the following summer. The cough is not severe, though sometimes paroxysmal, and expectoration is scanty, non-purulent, and may be confined to the morniug. Dyspnœa is not marked unless there is considerable emphysema. Acute exacerbations occur from time to time, and the tendency of the disease is to become worse from year to year, and to be more continuous, even persisting throughout summer.

In the *dry* catarrh, or *catarrhe sec* of Laennec, paroxysms of cough occur on the slightest provocation with the expectoration of small, hard pellets or without any expectoration. The patients are emphysematous.

The *diagnosis* is made by noting the long duration of the disease without impairment of the general health, its relation to season, and the absence of physical signs of involvement of lung tissue.

The physical signs of chronic bronchitis are those of bronchitis of the larger and middle-sized tubes. Large moist râles are more or less abundant, depending upon the degree of swelling of the mucous membrane and the quantity and fluidity of the secretion which is present. The respiratory murmur is roughened and less intense than normal.

W. Fox says that in chronic bronchitis there is commonly hyperresonance from coexisting emphysema, but under acute exacerbations the bases may be dull from congestion or œdema. Respiration is harsh, and in some cases of senile bronchitis expiration may be both prolonged and high pitched when other signs of dilatation of bronchial tubes are absent. The percussion note is clear.

The *sputa* of the severe forms of chronic bronchitis are usually copious and muco-purulent, the latter predominating. They vary in color from yellowish-white to ashen, greenish, or black when the lungs are anthracotic or collapsed.

The *subjective symptoms* of the patient consist, in ordinary cases, of a moderate amount of dyspnœa and tightness across the chest. At the onset of a fresh attack the symptoms may be those of acute bronchitis. The cough is paroxysmal, somewhat resembling that of whooping-cough, but without the characteristic whoop. It is usually severest on lying down and when rising in the morning.

The quantity and character of the sputa vary more than in acute bronchitis. Sometimes they are very copious, consisting of serum mixed with mucus, constituting *bronchorrhœa*. More commonly they are scanty, glairy, and tenacious.

Chronic bronchitis may be the result of repeated acute attacks, or, rarely, may follow one. It is frequently found in association with gout, chronic heart disease, chronic endarteritis, and Bright's disease, emphysema, asthma, and chronic alcoholism. It may interchange with other gouty affections, as articular inflammation or eczema, being relieved when the other manifestations are more marked. It also accompanies tuberculosis of the lungs. Climate and season have a marked influence; the disease is worse in damp, cold climates, and in the winter months.

Chronic bronchitis can be diagnosed from the cough of *aneurism* by the stridulous breathing due to paralysis of one-half of the vocal

cords, and by the local signs of a tumor of the vessel, which are in marked contrast with those of bronchitis. Other tumors may cause cough by pressure, but may be detected if the possibility of their existence is borne in mind.

4. Plastic Bronchitis is a form of bronchitis, usually chronic, the characteristic feature of which is the expectoration of fibrinous casts, which, when unravelled under water, are found to be solid casts of the smaller bronchial tubes. The casts are often tree-like in shape, showing that a bronchial tube and its smaller subdivisions had been occluded by the casts.

Persons of all ages are liable to it, but it affects males about twice as often as females.

The subjective symptoms are cough and dyspnœa; hæmoptysis occurs in about one-third of the cases (Biermer).[1] The cough occurs in paroxysms, which are frequent and severe; relief follows expectoration of the casts.

Hemorrhage may appear as streaks of blood upon the casts, or be considerable, and follow their dislodgment. The casts themselves are usually ejected coated with mucus, so that they appear as solid masses of sputum; their arrangement into cylinders may not be suspected until they are floated in water. The size of the cylinders varies from that of the little finger to that of a bodkin, but they do not often exceed the size of a goose-quill. The larger casts may be hollow, but the smaller ones are solid, and are arranged in layers. They are whitish or grayish in color, and firm in consistence, but become softer as the disease improves. Microscopically, the casts are nearly structureless, consisting of a fibrillated base, scattered with pus and mucous corpuscles, a few gland-cells, and occasionally blood-cells in the outer layers. Charcot-Leyden crystals and Curschmann's spirals are found.

The *acute* form is rare, and out of ten cases accepted by Biermer, six proved fatal. The disease begins with fever, dyspnœa appears early, severe paroxysms of cough occur, sometimes hemorrhage. Death results from asphyxia. Grave symptoms are excessive dyspnœa with scanty expectoration and drowsiness. Copious expectoration is a favorable sign.

The duration of the chronic form is very variable, some cases lasting a number of years; but it is not as a rule dangerous to life, nor does the general health suffer much.

The Physical Signs. The casts obstruct the bronchial tubes. There is lessened amount of air entering the part, hence there is lessened fremitus and diminished respiratory murmur over the portions of lung supplied by the obstructed tubes. If collapse ensues there will be dulness on percussion; if the casts are dislodged, the murmur becomes normal, or but slightly roughened. Over unaffected portions of the lung resonance is clear or exaggerated, and respiratory murmur unaltered.

Fuller says (quoted by Peacock: *Diseases of Chest*) that the upper portions of the lungs are oftener affected than the lower portions.

5. Fœtid or Putrid Bronchitis is the name applied to the condition in which the sputa have a highly offensive odor and are copious

[1] Virchow: Handbuch der spec. Path. u. Ther., Bd. v. Abth. 1.

and semi-putrid. The odor is said by some to be due to microscopic sloughs, and by others to a special bacillus.

Putrid bronchitis may accompany (1) dilatation of the bronchial tubes; (2) chronic pneumonia; (3) phthisis, or (4) empyema with a fistulous communication with a bronchus; or (5) it may occur independently. The subjective symptoms are cough, irregular fever, and occasional chills. The physical signs are those of chronic bronchitis, or of bronchitis and of the conditions with which it may be associated (*q. v.*). From *gangrene* it is diagnosticated by absence of physical signs of disintegration of lung tissue and by the absence from the sputum of fragments of lung tissue and elastic fibres. Nevertheless gangrene of the lung may be the final result of putrid bronchitis.

The sputa of fœtid bronchitis has an odor of gangrene or fæces. On standing they separate into three layers. The upper one consists of a greenish fluid or frothy layer; the second is sero-albuminous, and the third a thick granular deposit in which are small masses the size of peas (Dittrich's plugs) and flakes consisting of granular detritus and containing fat crystals and bacteria, the oïdium albicans, and crystals of leucin and tyrosin (Wilson Fox). (See Sputum.)

Specific Bronchitis.

In addition to the bronchitis that attends the infectious disorders mentioned above, three forms are seen of an infectious nature which are properly classified among the infectious disorders. It is proper to refer to them now, as bronchitis is usually the most pronounced local manifestation. They are influenza, whooping-cough, and hay fever. The last only will be spoken of at present.

Hay Fever.

Hay fever is a specific catarrh of the respiratory passages, caused by the pollen of certain plants, principally the grasses. The attack begins with itching, burning, and lacrymation of the eyes, and pain in the brow or eyeballs. Subsequently there is itching or pricking of the nasal mucous membrane, frequent sneezing and an irritating watery discharge. The mucous membrane of the nose is red and swollen. A similar condition obtains in the throat when that is affected. If the disease attacks the bronchial mucous membrane a bronchitis is set up, which differs, if at all, from an ordinary bronchitis in being more persistent and in being attended by greater dyspnœa, with asthmatic attacks.

Bronchiectasis.

Dilatation of the bronchi occurs secondarily to affections which tend to weaken the walls of the tubes and to lessen their elasticity. Hence it is found in chronic bronchitis with emphysema, in chronic phthisis, in catarrhal pneumonia in children, in chronic obstruction from external pressure or foreign bodies (see Obstructions). It also occurs when the

lungs contract in fibroid pneumonia or pleural thickening. It occurs in two principal forms: the *simple*, in which the affected tubes are uniformly dilated; and the *saccular*, in which larger or smaller pouches are formed. It is commoner in males than in females, and probably begins most frequently in adult or middle life. One lung only is affected in about one-half the cases, and when both lungs are affected (chronic bronchitis and emphysema) it is not often to the same degree.

The subjective symptoms consist of cough, expectoration, and a variable amount of dyspnœa. Eventually there may be some loss of flesh and strength.

The *cough* is usually paroxysmal. It may occur only in the morning after the dilated tube fills. It may follow change in position. A paroxysm is followed by copious expectoration, sometimes amounting to a pint and a half in twenty-four hours. It is grayish-brown and muco-purulent, faintly or extremely fœtid. The *sputa* contain mucus, pus, casts of the tubules, and various salts. Charcot-Leyden and fatty crystals, vibrios, leptothrix, and bacteria (Fox) can be found on microscopic examination. Elastic fibres are found only if the tubes are ulcerated. In a conical glass it separates in three layers—a frothy brown top, a thin mucoid layer in the middle, and below a granular layer. Hemorrhage is rare, but may occur even when tubercle is absent.

Dyspnœa is not usually severe except when the dilatation is complicated by disease of the heart or lungs, or during an acute attack of bronchitis.

The *physical signs* differ according to the extent of the dilatation and its kind. In simple dilatation there may be nothing different from the signs found in chronic bronchitis, except a tendency to more bronchial respiration, with râles having a metallic quality. Percussion will vary according to the degree of alteration of the lung tissue surrounding the affected bronchi and according to the extent of the dilatation and its nearness to the surface. In the simple forms the percussion note if altered is somewhat less resonant and higher in pitch, whereas in saccular dilatations favorably situated for percussion the note is tympanitic if the pouch is empty. On auscultation in simple dilatation the breathing approaches the bronchial, and is accompanied by bronchial râles. In saccular dilatation the sounds are practically those of a cavity, respiration varying from bronchial to amphoric. Vocal resonance and tactile fremitus are generally both increased, but the latter may be diminished.

The *diagnosis* of simple dilatation from chronic bronchitis may be impossible, but copious and fœtid expectoration indicates it. The diagnosis of the saccular form from tuberculosis of the lung with cavity is difficult. Wilson Fox says the severer cases are usually associated with consolidation of the lung or with tubercle; but even without the presence of the latter they often present phthisical symptoms—retraction of the chest, with the physical signs of excavation, pains in the side, hæmoptysis, pyrexia, nocturnal perspiration, and diarrhœa—which may all coexist with only an induration of the lung and dilatation of the bronchi. The diagnosis must be made by noting the persistency of the physical signs, which change but very little and are not progressive

as are those of tuberculosis; the protracted course of the disease; the character of the sputum; and the comparatively slight impairment of the general health.

Obstruction of the Bronchi.

Obstruction may be produced by causes external to the tubes, or by internal causes, *i. e.*, may be due to compression or to constriction.

Compression may be by tumor, enlarged glands, aneurism, hydatid cyst, mediastinal abscesses, and long-continued pleural effusions and goitre.

Constriction may be produced by swellings of the mucous membrane, by polypoid growths, or by growths forming in the lung and extending into the bronchi. Cicatrices may be produced by syphilis, tubercle, or by pleural thickenings.

The symptoms depend upon the size of the tube and the degree of stenosis. When *small areas* are affected there may be no demonstrable physical signs, because the lung around the affected area becomes emphysematous. When *large areas* are affected, percussion often continues resonant, but its limits are said to be less influenced than in health by forced inspiration and expiration. The breath-sounds are weakened, and vocal resonance and fremitus are diminished in intensity and may be absent. Sibilant and sonorous râles may be heard at the site of the obstruction, and fremitus may be felt over the corresponding area. Dyspnœa is proportioned to the stenosis and the size of the tube occluded.

Asthma.

Asthma is a chronic disease depending upon spasmodic narrowing of the bronchial tubes, and characterized by paroxysmal attacks of dyspnœa, diminished respiratory movement of the chest, prolonged expiration attended by a wheezing sound and sibilant râles, and ending abruptly with the expectoration of a tenacious mucus. The attack may be limited to a single night, or may be prolonged for days, with nocturnal exacerbations.

Premonitory symptoms are said to occur in about one-half the cases. These are for the most part nervous, such as headache, neuralgia, irritability of temper, vertigo, drowsiness. Hyde Salter found that there were premonitory symptoms in 111 out of 226 cases collected by him. In 63 they were nervous, in 8 there was profuse diuresis, and in 14 they were connected with the digestive system.

The attack itself usually begins during sleep, and often at a regular time. It may, however, begin during the day, and at a certain hour, independently of sleep. The onset is manifested by tightness across the chest and more or less difficulty in breathing. This dyspnœa increases rapidly and often reaches an extreme degree. The face becomes pale and anxious, and may be moistened with a cold perspiration; the lips are dusky from deficient oxygenation of the blood. The patient feels smothered, and makes frantic efforts to get his breath, rushing to an open window, no matter how cold the weather, or if unable to leave

the bed, sitting up with the hands pressed upon the bed so as to give purchase to the accessory muscles of respiration. Notwithstanding great respiratory efforts are made, the chest moves but little, because the lungs are already distended to the extent of a full normal inspiration. The patient is unable to expel the contained air owing to the spasm of the bronchial tubes.

The frequency of respiration is diminished, sometimes to one-half the normal; the rhythm also is altered, inspiration being short and gasping, and followed without pause by expiration, which is much prolonged and accompanied by a wheezing sound audible to bystanders.

There is an increased amount of air in the thorax, and inability to remove it. The chest is enlarged—barrel-shaped—the movement is lessened and strikingly out of proportion to the efforts. The diaphragm is lowered.

The *physical signs* are hyper-resonance on percussion; on auscultation, faint, short inspiration, prolonged expiration, and sibilant and sonorous râles, more marked on expiration.

The duration of an attack of asthma varies from half an hour to a day or two. In patients with chronic bronchitis it may be prolonged for a week or two, with remissions during the day. It may subside abruptly or by degrees.

Subsidence of an attack is marked by expectoration, the sputa having special characteristics (see under Sputum). At first it is made up of rounded gelatinous masses which, when unfolded in water, are made up of spirals. Later, it becomes muco-purulent.

No duration can be set down for the disease itself. It may be said that the earlier the age at which it begins the better the prospect of ultimate cure. If a cause can be discovered and its operation avoided the prospect of a cure is increased.

The causative factors in asthma are various. About twice as many males as females are affected, and there is a marked hereditary tendency in some families. There is probably some special peculiarity in asthmatic patients, but just what it is has not been determined. It may reside in the lungs, and may be part of a general constutional irritability (Salter). Bronchitis, emphysema, and heart disease act as causes, and so do syphilis, malarial poisoning, and chronic Bright's disease.

Diseases with Increased Amount of Air.

Emphysema.

Emphysema consists in an "excessive, permanent, and unnatural distention of the air-cells," or in "extravasation of air into the interlobular or subpleural cellular tissue." (Laennec.)

Emphysema may be unilateral or bilateral. Local and unilateral forms are usually compensatory. Bilateral emphysema may be hypertrophic or atrophic.

It is more common in men than in women. Its symptoms are more common in childhood and after middle age. Two factors are essential in its causation. First, defective development of the elastic tissue of

the lungs. Second, increased intra-alveolar air-pressure. The latter is due to a number of causes. In childhood no doubt nasal and naso-pharyngeal obstructions are operative. In adults, occupations which necessitate continous and severe muscular effort, especially if coupled with forced expiration with closed glottis, act as causes. Such occupations are blacksmithing and playing upon wind instruments of music. Diseases which compel much coughing or respiratory effort, such as chronic bronchitis and whooping-cough, act in the same manner. Chronic mitral valvular disease and the lessened elasticity of the lung tissue which come with advancing age both favor congestion of the lung, and thereby predispose to emphysema. The disease is hereditary. Several members of a family are affected. It occurs in many in childhood, is in abeyance in adult life, and reappears in old age.

Symptoms. The prominent symptoms in hypertrophic emphysema are *dyspnœa*, *cyanosis*, and *cough*, with expectoration from associated bronchitis. There is no fever. The dyspnœa is proportioned to the degree of emphysema present, and is aggravated by the coexistence of bronchitis, asthma, and eccentric hypertrophy of the right ventricle, which are very frequent complications in cases of long standing. When the degree of emphysema is only moderate, dyspnœa is not complained of except upon climbing or walking briskly, or after a hearty meal. But when the degree of emphysema is great, dyspnœa is constant; it interferes with all exertion, frequently necessitates orthopnœa, and prevents continuous speech; such patients speak in broken sentences or syllables.

Cyanosis is marked. The livid lip is common in the asylums for old men. The extremities are also dusky, and the blueness is general in severe cases. This cyanosis, the round shoulders, and the drawn, chronically anxious expression, if I may so term it, make it easy to pick out the emphysematous subjects in a ward of chronic cases.

The rate of respiration is not accelerated, and may be diminished in frequency. It is often accompanied by wheezing when chronic bronchitis coexists.

The cough varies greatly in frequency. It may be altogether absent, since its presence simply indicates an associated bronchitis. This bronchitis may be present only in the winter for a long time. It may arise on changes of the weather. Finally it becomes chronic. The expectoration is that of chronic bronchitis (*q. v.*) It is rarely stained with blood.

The *physical signs* of emphysema depend upon its degree, and whether complicated with chronic bronchitis or not. *Inspection:* In well-marked cases the chest is barrel-shaped (see under Inspection). There is little movement of the chest in respiration, because the lung is already in a condition of full inspiration (expiratory dyspnœa). *Vocal fremitus* and *resonance* are usually diminished. *Percussion:* The percussion note is abnormally clear, and may even be tympanitic. Hyper-resonance is typical of the disease. When the distention is extreme the note may be woodeny. (See Fig. 36.) The lungs are enlarged. The heart dulness becomes obliterated by the overlapping lung. The upper margin of the liver falls one or two interspaces

below the normal. The resonance extends higher above the clavicles than normal.

On *auscultation* the inspiration is found to be distant and feebler than normal, while the expiration is prolonged, and may become three or four times the length of inspiration. Grazing or rubbing sounds have been described, and attributed to the friction of distended vesicles against the pleura. Other adventitious sounds are due to an associated bronchitis, pleurisy, or tuberculosis. But bronchitis is such a common accompaniment of emphysema that the râles of the former become almost symptomatic of the latter. Their character in emphysema does not differ from that in chronic bronchitis (*q. v.*)

The Heart. The apex beat is absent. There is epigastric pulsation or systolic shock. The normal area of heart dulness is encroached upon by the distended lung, and the heart itself is pushed to the right, the apex beat frequently being at the xiphoid cartilage. If the emphysema attain a very high degree there may be no perceptible dulness except on very strong percussion over the cardiac region. The heart sounds appear feebler and more distant than normal. The right ventricle becomes dilated and hypertrophied, as the result of the pulmonary congestion produced by emphysema. The pulmonary second sound is accentuated. A tricuspid regurgitant murmur is heard. Venous congestions are common in the later stages. Albuminuria is common. Œdema of the feet and limbs may occur, but general anasarca is rare.

The general health suffers by loss of strength and capacity for physical and mental work, rather than by loss of flesh. The patients are large chested, stoop-shouldered, and short-breathed, and have an anxious expression of countenance. The face is of a dingy pale color, but becomes bluish on exertion.

Diagnosis. This is based upon the history (heredity, occupation, long duration), the occurrence of dyspnœa and cyanosis, and of winter cough or chronic bronchitis, and the physical signs.

Emphysema can be distinguished from *pleural effusion* and an *aneurism*, which may cause dyspnœa, by the universal hyper-resonance on percussion. Pleural effusion, which also causes bulging, is usually unilateral, and the percussion note over it is flat. There is diminution of areas of dulness about the heart and aorta in emphysema.

Pneumothorax, which most resembles emphysema, develops suddenly, affects one side, and has a hollow, tympanitic note on percussion. The succussion splash, metallic tinkling, and coin test have no counterpart in emphysema; moreover, the antecedent history and mode of development are different.

Atrophic Emphysema is due to the degeneration of age. The lung is reduced in size. The diameters of the chest are lessened. The ribs are oblique. There is atrophy of the chest muscles. The patients have dyspnœa. There are other signs of senility.

In *interlobular emphysema* the physical signs are the same as those of vesicular emphysema, but it develops suddenly and is liable to be followed by emphysema (intercellular) of the neck, which on palpation gives a peculiar crepitation. The friction sound and crackling which

have been described as occasional adventitious sounds in vesicular emphysema are more commonly heard in the interlobular form.

It is caused by rupture of the air-cells, and hence occurs in diseases in which a great strain is put upon them—especially, therefore, in whooping-cough, but also occasionally in pulmonary hemorrhage and pneumonia; violent coughing and laughing, and great straining, as in child-labor, are capable of producing it.

Diseases with Diminished Amount of Air.—The Consolidations.

Congestion of the Lungs.

Active Congestion. In *active congestion* there is increased amount of blood, which diminishes the air-space by encroachment and causes more or less consolidation. The signs of that physical condition are present—increased fremitus, impaired resonance or dulness, and bronchial breathing. The signs are observed on both sides, usually the bases. Dyspnœa, cough, and frothy, bloody expectoration attend the fluxion. Cases have not been reported in which bacteriological examination of the sputum was made. Of course the *micrococcus lanceolatus* is not found.

If the above signs and symptoms develop suddenly—within twenty-four hours—a fluxion to the lung has in all probability taken place. If the patient is a subject of heart disease, or if he has been exposed to and has inhaled hot vapors or irritants, the probability of fluxion is increased. The occurrence of the symptom fever would point to *pneumonia* as the cause of the objective and subjective symptoms.

Passive Congestion. The physical condition that results is consolidation. The bronchial mucous membrane is also congested. On account of the former there is slight dulness and feeble or bronchial breathing; on account of the latter, abundant râles. The affection is bilateral and usually confined to the bases, and of these, to the posterior portions. It is also secondary. *a.* Mechanical congestion occurs when the flow of blood to the heart is obstructed, as in organic valvular disease or insufficiency. Rarely, the pressure of tumors on the pulmonary veins acts in a similar manner. *b.* Hypostatic congestion occurs in fevers, as protracted typhoid, and in prolonged general exhaustion or adynamia. Ascites or other affections below the diaphragm, which lessen the respiratory excursion, cause this form. *Dyspnœa*, *cough*, and expectoration, with blood-stained sputum, are common. The sputum contains alveolar cells, but no micro-organisms.

Œdema. The air-cells and alveolar walls are filled with serous exudation, as in œdema of the skin. It is frequently due to the weakness of the heart, which occurs at the end of long-continued diseases of an exhaustive nature, particularly if stress is thrown on the heart. It occurs, therefore, in the terminal stages of chronic Bright's disease, of organic heart disease, of the anæmias and cachexias. Both congestion and œdema occur in cerebral affections.

Symptoms. They are those of congestion in a more aggravated degree. Dyspnœa, cough, and the expectoration of large quantities of

a sero-mucoid fluid are seen. The diagnosis is based upon the results of physical examination and the history of the presence or absence of the above causal factors.

Pulmonary Embolism and Thrombosis.

Pulmonary embolism consists in plugging of the pulmonary artery or its branches by coagula formed in the right heart or in the veins. The symptoms depend upon the size of the occluded vessel and upon the nature of the embolus, *i. e.*, whether septic or not. If the artery itself is plugged, death takes place suddenly, or after a short interval, with symptoms of syncope or asphyxia.

Symptoms. If a large branch is plugged, the first symptom is generally intense dyspnœa, which may amount to an agonizing craving for air. Pain in the chest, which may or may not be acute, is complained of and may be referred to the seat of the embolus. Cough is not a common symptom, and may be altogether absent. The breathing is considerably altered; it is usually increased in frequency, and may be much hurried; it may or may not be shallow, and while the patient can take a deep inspiration, it does not give relief to his dyspnœa. At times it is irregular and gasping.

The face is pale, or may be cyanosed, and is apt to be bathed in perspiration. The veins are swollen and prominent. The heart's action is irregular and may be tumultuous. Exophthalmos has been observed. The temperature falls below normal, but a febrile rise may occur later. The intellect is unclouded.

The physical signs are indefinite. The respiratory murmur is roughened and exaggerated in most, but not in all cases. Fox states that râles are very rarely heard. Collapse, œdema, and bronchitis are possible results. A systolic blowing murmur may be heard over the heart and pulmonary artery, and in protracted cases albuminuria and œdema may be met with.

When the embolus is septic, a *septic pneumonia* or *metastatic abscesses* are probable results in cases not immediately fatal.

When the emboli produce *hemorrhagic infarcts*, the symptoms are milder and consist principally in dyspnœa, pulmonary hemorrhage, and palpitation. The onset is sudden and accompanied by a fall in temperature. The physical signs indicate consolidation, if the pneumonic or infarcted area is of moderate size. It may be discovered at the root of the lungs in the inter-scapular region.

Hæmoptysis is a common symptom when the embolus has arisen in the heart. The amount of blood varies from a copious expectoration to the rusty sputum seen in pneumonia, but may persist for weeks. *Pleurisy* and pleural effusion are frequent complications; chills occur sometimes, and *pneumonia*, with corresponding rise of temperature, may develop.

The most important points in diagnosis are the sudden onset of the dyspnœa and other pulmonary symptoms, and the detection of a condition which would give rise to emboli, such as puerperal fever or heart disease.

Pneumonia.

Acute pneumonia, croupous or lobar pneumonia, is an infectious inflammatory disease excited by the micrococcus lanceolatus (diplococcus pneumoniæ, pneumococcus) involving the vesicular structure of the lungs, and followed by choking of the alveoli with the products of inflammation.

Symptoms. *Mode of Onset.* The invasion of pneumonia is usually sudden, and is marked by a *chill.* The temperature rises rapidly and may reach 104° to 105° in the first twelve hours after the chill. With the fever, the patient complains of severe headache and *pain* in the side, and has a short, quick cough and sometimes vomiting. The pulse is accelerated moderately, and the respiration either is or soon becomes very frequent. The face is apt to be flushed, and there may be a circumscribed red spot on the cheek. The skin is hot and dry. On physical examination, within the first twenty-four hours, a small patch of consolidation is detected, which may subsequently extend over a large area.

While this is the picture of an ordinary pneumonia in its early stage, all cases are by no means so clear. In some the course resembles that of a general fever in which the pulmonary disease is a local manifestation. In such cases there may be prodromata, consisting of headache, general malaise, a slight bronchitis, and digestive disturbance. Then follows the chill. *Central pneumonia.* The fever may be high for several days before there is any discoverable consolidation of the lungs, and during this time cough be wholly or almost wholly absent. The respirations increase gradually in frequency, and finally a well-marked pneumonia can be made out. It is customary to account for these cases on the supposition that pneumonia developed in the interior of the lung and consolidation gradually extended to the surface. In some cases the patient presents no more definite symptoms for three or four days than high fever, intense headache, and moderately accelerated respiration.

Later Stages. At the end of forty-eight hours, or at the most, of four days, the patient is found lying in bed in the dorsal position, or on the affected side. The face is flushed, the countenance anxious, the respirations hurried, the alæ nasi play vigorously. The temperature varies little from the first day's rise; the chest pain has been relieved, the short, dry cough is now attended by viscid expectoration. The respiration continues hurried, the pulse full and bounding. During this time the physical signs of consolidation continue and increase.

After a period of five or ten days, the termination takes place by crisis. Previous to crisis, the pain in the chest lessens, the cough becomes looser, and the expectoration more free, but the other symptoms persist. In addition, in some cases, delirium occurs, the pulse softens and becomes dicrotic, the urine becomes albuminous.

Respiratory Symptoms. Chest pain, cough, hurried respiration of a peculiar type, and expectoration are characteristic. The *chest pain* is sharp and stabbing or lancinating. It is increased by breathing. It is seated about the nipple or in the *axillary region*, at the angle of the

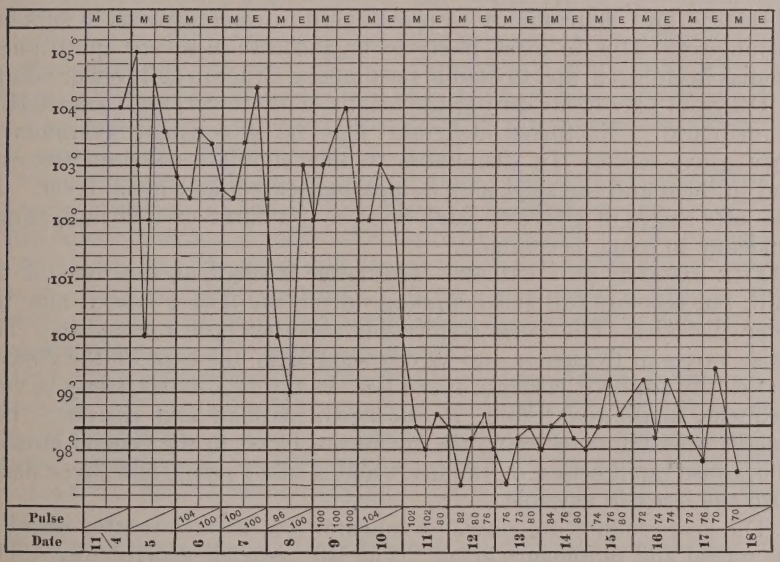
scapula or complained of below the diaphragm. Its seat always indicates the side affected. *Cough* is short and dry, smothered and painful; it soon becomes softer and painless as the expectoration becomes free. It may be absent in the feeble, in the aged, in alcoholic subjects, or in persons with brain disease, including insanity.

A characteristic symptom of pneumonia is the increased frequency and the type of the *respiration*. The frequency in adults reaches 40, 50, or even 60 per minute, and in children 80 and 100 are not very uncommon.

The pulse, on the contrary, does not increase in frequency in the same proportion; hence, the normal ratio of respiration to pulse of 1 to 4 ceases, and becomes 1 to 3 or 1 to 2.

Inspiration is short, expiration quick and attended by an expiratory noise or grunt. The long pause may take place after inspiration instead of expiration. In children both are so short that unless the epigastrium is inspected it may be difficult to distinguish the two.

FIG. 56.



Pneumonia. Sudden rise; termination by crisis. Pseudo-crisis also seen.

In ordinary cases which run a normal course, following the cough there is expectoration, which is at first viscid mucus, but gradually becomes reddish-brown from admixture of blood—the *rusty sputum* of pneumonia. This sputum is characteristic, almost pathognomonic. It is expelled with difficulty from the mouth, clinging to the lips, or if a male, to the mustache, when present. It cannot be removed from the spit-cup when turned over. It continues to be rusty, and as the crisis approaches becomes purulent and is discharged with ease. In typhoid pneumonia, it looks like prune-juice (see sputum). It contains blood, alveolar epithelium, the specific micrococcus, and later, pus and small fibrinous casts.

The Fever. The chill that precedes the fever is pronounced and always a warning to look for a pulmonic inflammation. If in children a convulsion is rarely absent in frank pneumonias. During its occurrence, the body temperature rises. The temperature in twelve hours reaches 104° or 105.° It remains at this point, obeying the laws of diurnal variation. The hot dry skin, the parched lips, the dry tongue, the thirst, the anorexia, the hurried breathing, the occasional delirium, the loaded urine attest its presence. At the end of the third or more frequently the fifth, seventh, or ninth day, *crisis* takes place; the fall is abrupt and the normal or a subnormal temperature may be reached in from five to fifteen hours. *Pseudo-crisis*, as the accompanying chart indicates, may precede true crisis by twenty-four or forty-eight hours. The decline may take place by lysis, however. Protracted fever indicates delayed resolution or the occurrence of a complication.

Cerebral Symptoms. In some cases, especially in children, the onset of the disease may be marked by a convulsion. This is said to occur more frequently in apical pneumonias than in pneumonias of the base. Headache and delirium are so pronounced in some cases as to simulate meningitis. This is most likely to be the case in severe apical pneumonias in children, and in double pneumonia, either in children or adults.

Delirium may occur during the height of the fever, and occasionally is maniacal. Nocturnal delirium may be a constant symptom in very grave cases. In drunkards it may simulate delirium tremens, and in them and the aged may be pronounced without much fever. In the later stages of grave or fatal cases, a low form of delirium, with a tendency to coma, is common.

The Heart and Pulse. The latter is small at the time of the chill, but becomes full and bounding during the fever; later it may become dicrotic. The pulse-respiration ratio has been referred to. The pulse varies in frequency and in character with the type of the disease. In healthy adults it is rarely over 110. In the debilitated it may be very frequent, small, and feeble; in the aged, frequent and dicrotic. Extensive consolidations lessen the amount of blood in the general circulation, cause rapid action of the heart and a small pulse, and favor death with the heart in asystole.

The heart sounds are clear. A murmur low in pitch is often heard in the mitral and pulmonary areas. The left ventricle acts forcibly. The pulmonary second sound is accentuated. If dilatation and failure of the right heart take place, the area of dulness may extend beyond the right edge of the sternum, an epigastric impulse be noted, turgescence of the veins in the neck become marked, but above all, the previously accentuated pulmonic second become weak or disappear.

Gastro-intestinal Symptoms. Vomiting frequently occurs in children at the onset, and both in them and in adults may persist and mask pulmonary symptoms. The appetite is lost. The tongue is furred. It may become dry and brown. The bowels are constipated except when complications occur. The *spleen* is enlarged.

Cutaneous Symptoms. Herpes on the lips, the nose or the genitals is of common occurrence. Sweating occurs with the crisis, or if heart failure is imminent.

The Urine. The urine is scanty and high-colored, and may contain a small amount of albumin. In some cases the chlorides are found to be absent. This is determined by acidulating the urine with a drop or two of nitric acid, and then adding one or two drops of a ten per cent. solution of silver nitrate. If chlorides are present a heavy white cloud of chloride of silver is thrown down. The chlorides are not invariably absent, or even diminished in pneumonia, hence their reappearance, which is said to indicate beginning convalescence, loses its value as a prognostic sign.

Physical Signs. *Consolidation.* Diminution in the amount of air, increase of solid contents. On *inspection,* diminished movement. If extensive consolidation, enlargement of the affected side. On *palpation,* inspection confirmed and increased vocal fremitus discovered. Both are more marked at the height of consolidation. *Percussion.* In first stage, impaired resonance or Skodaic resonance. In stage of hepatization, dulness or flatness, but without any wooden quality or marked resistance. *Auscultation.* In the early stage, that of congestion, the respiratory murmur is suppressed and crepitant râles are heard at the end of inspiration. On full inspiration or after a cough a broncho-vesicular respiration is brought out. When consolidation has taken place the respiratory murmur is bronchial. Râles, if present, are moist subcrepitant râles from associated bronchitis, or a few crepitant râles may still persist, and a friction sound be heard.

When resolution sets in, the crepitant râle reappears, quickly followed by moist subcrepitant râles heard both on inspiration and expiration, while dulness gradually yields to impaired resonance. The respiration loses its bronchial character and again acquires a vesicular element before becoming completely normal. It may be a week or two, or many months, even in uncomplicated cases, before the percussion note becomes perfectly clear and râles wholly disappear.

Duration and Course. The duration of the disease is from one to two weeks. It may subside by crisis on the third, fifth, seventh, or ninth day, or gradually by lysis. Crisis is marked by a critical sweat, a copious discharge of limpid urine, or sometimes by a few loose movements of the bowels, accompanying a fall of temperature to or below normal.

Instead of clearing up, the pneumonia may progress to suppuration, abscess, or gangrene. These conditions can be made out by the character and range of temperature, the general condition of the patient, the sputum, and the physical signs. Termination in abscess or gangrene is rare.

In cases proceeding to a fatal issue the strength fails, respiration becomes more labored, and expectoration increasingly difficult. The number of respirations frequently diminishes, but the pulse continues frequent and often becomes small and irregular. Physical examination shows diffuse bronchitis with œdema. The heart's action is irregular and rapid. The sounds are weak and feeble; the first becomes short and snappy like the second, and later both are weak or indistinct. Death may occur abruptly from convulsion, or more frequently from the development of asphyxia, due to œdema of the lungs, which in turn set in

on account of weakness of the heart or the development of heart-clot from cardiac asystole.

VARIETIES. *Migratory pneumonia.* Sometimes, with the reappearance of abundant râles and increased expectoration, the fever continues high, the patient is disinclined to take food, has a dry, brown tongue, and is often delirious. In such cases the pneumonia is probably extending in the lung already involved, or has attacked the other lung.

Typhoid pneumonia is an unfortunate name for an adynamic form of the disease with typhoid symptoms. If it arises in the course of, or complicates, low fevers, it is usually of the typhoid type; but it occurs also in those much exhausted, in depraved health, or exposed to unhygienic surroundings. It is found also in cases of septicæmia, in Bright's disease, in drunkards, and in the negroes in the southern part of the United States.

The characteristic features of this form of pneumonia are the great physical prostration and the weak heart-action. The fever is high, the respiration and pulse frequent, and delirium and vomiting are more frequent than in the ordinary form. The skin sometimes has a dusky hue; the tongue is heavily coated, or may be dry and brown, and sordes collect on the teeth. The sputa may be rusty, and sometimes pure blood is expectorated. The disease may prove fatal rapidly, or may linger for a long time, the patient only gradually coming out of a low typhoid state. It is always dangerous.

Bilious pneumonia is the name given to a type of pneumonia occurring in persons laboring at the same time under malarial poisoning. The initial chill lasts longer, and the pain in the side, from coincident pleurisy, is more marked than in ordinary pneumonia. The fever is more remittent, and jaundice and vomiting are present.

DIAGNOSIS. The diagnosis is based upon the aggregation of special symptoms. The mode of onset, the chill, the course of the fever, the pain in the chest, the cough, the peculiar expectoration, the dyspnœa, the abnormal pulse-respiration ratio, the peculiar character of breathing, and the physical signs are common symptoms. It must be remembered that in children, in the aged, in drunkards, in cases of chronic disease, the type is deviated from. In drunkards cerebral symptoms are more marked. In children the cerebral symptoms are more prominent, the expectoration often absent. In the aged, the cough, the expectoration, and the fever are not pronounced; the former may be absent; the onset is insidious. The same onset and course occur in wasting diseases, as cancer, phthisis, Bright's disease, diabetes, and organic heart disease. It must be remembered that in this class of cases a small patch of pneumonia, difficult to determine on physical examination, may be attended by the gravest general symptoms. In all of the above cases, if there is *fever* without cause, although no pulmonary symptoms are present, the lungs must be examined repeatedly. In many of such cases the physical signs are obscured because respiratory action is enfeebled by the primary condition.

Pneumonia must be distinguished from other acute inflammatory affections of the lung and pleura and from acute tuberculo-pneumonic phthisis. The evidence for each is considered in the respective sections.

To distinguish pneumonia from pleurisy with effusion the aspirator must be used.

Bacteriological Diagnosis. Staining and microscopical examination of the sputum reveals the characteristic micro-organism. Care must be taken to secure sputum from the lung. By inoculation of rabbits with the sputum the disease is readily reproduced. The organism is not readily found in the blood.

Complications. The complications which occur in the course of the disease and modify the clinical picture and obscure the diagnosis are: pleurisy with serous or purulent exudation, pericarditis, endocarditis, meningitis, and jaundice.

Broncho-pneumonia, or Catarrhal Pneumonia, is a pneumonia occurring secondarily to bronchitis, and is characterized by the development of areas of consolidation in both lungs and the persistence of a bronchitis of the middle-sized or smaller tubes. In proportion as the areas of consolidation are large, the symptoms and physical signs approach those of lobar pneumonia. It is more common in children and in debilitated persons. It is the chief form in infants. 1. It is frequently secondary to measles, diphtheria, scarlet fever, and pertussis. 2. As aspiration-pneumonia, it occurs when food, septic particles, blood or tissue enter the lungs during the loss of sensibility of the larynx in apoplectic, uræmic or other forms of coma, and in operations about the upper air-passages and mouth. It is a fatal complication of tracheotomy. 3. It is frequently of tuberculous origin.

Catarrhal pneumonia, except the aspiration form, develops gradually, and it may not be easy always to mark the point at which the bronchitis which precedes merges into pneumonia; but as a rule there is more or less chilliness (rarely a decided chill), and an access of fever. There is usually greater prostration in proportion to the amount of pneumonia present than in the lobar form. The pulse is more frequent and more likely to be feeble. Cough and expectoration are marked symptoms. The sputum is tenacious and glairy, not rusty. Dyspnœa is more extreme than in lobar pneumonia. The respirations are excessively rapid—sixty to eighty per minute; cyanosis rapidly ensues. The finger-tips become blue, the face dusky. The fever does not rise as high as in the lobar form. At first the skin is hot and dry; later it becomes cool and clammy, and in the tuberculous form sweats are common. The duration of the disease is usually much longer than in lobar pneumonia.

The physical signs are those of bronchitis, with here and there larger or smaller areas of consolidation, over which the râles are finer and closer set; the percussion note is dull, and the respiratory murmur bronchial or broncho-vesicular. Areas of collapse and portions more or less œdematous combine to make up the complex of physical signs. While both lungs are affected they are not usually affected to the same degree. It is said that the apices are more prone to involvement in this than in the lobar form; and some writers (Osler) look upon it as a form of phthisis.

In the common form seen in infants the symptoms of asphyxia set in at variable periods in the course of the disease. General cyanosis

supervenes. Stupor sets in, the hurried respirations grow shorter and more gasping, the pulse becomes excessively rapid and feeble, the extremities cool and clammy; with the stupor the cough lessens and the breathing becomes more shallow. The lungs fill up with fluid mucus, and the child drowns in its own secretions, or cardiac paralysis sets in after dilatation of the right heart.

Diagnosis. The affection is distinguished (1) by its pathological antecedents and causal relations; (2) its gradual onset; (3) its distribution in both lungs; (4) the preponderance of physical signs of bronchitis over those of consolidation; (5) the extreme dyspnœa and cyanosis with a lower temperature than in lobar pneumonia; (6) the onset of carbon dioxide poisoning; (7) the long duration and gradual decline. The tuberculous form is distinguished by (1) the history of exposure to infection or of a focus of infection in the body, glands, or joints; (2) the longer course; (3) delayed asphyxia; (4) rapid emaciation; (5) profuse sweats; (6) physical signs of consolidation and subsequently of cavity at the apex; and (7) absolutely by tubercle bacilli in the expectoration coughed up or vomited. I have seen a child aged fifteen months, of a tuberculous mother, completely recover. The tuberculous form is common in colored infants.

Chronic Interstitial Pneumonia.

Cirrhosis, fibroid phthisis, and chronic interstitial pneumonia are names given to a condition of chronic induration of the lung caused by an interstitial overgrowth of fibrous tissue. Obliteration of the air-vesicles and contraction of the lung result from the overgrowth. The bronchi are frequently dilated, and cavities and gangrene may occur. The disease is rare except as the result of tubercle, but it may follow pneumonia and pleurisy, and it is said to be caused by the inhalation of fine particles of steel or cotton. Pneumonokoniosis is the term, first employed by Zenker, for the chronic interstitial pneumonia from the inhalation of dust.

Physical Signs. *Inspection.* The disease is unilateral. The chest wall is *retracted.* The ribs are drawn together so that the interspaces are obliterated. The shoulder is drawn over the sunken thorax. The spinal column is curved. The heart is displaced. It is drawn toward the affected side. If the right lung is the seat of disease an impulse is seen to the right of the sternum; if the left, the præcordial area of impulse is increased and extends upward. There is no expansion whatsoever (immobility) of the affected apex or base. The healthy lung is the seat of compensatory emphysema.

Palpation. Inspection is confirmed. Fremitus is increased, especially at the apex. At the base pleural thickening lessens fremitus.

Percussion. The physical signs show increased density of lung tissue, with dulness on percussion, or, over a dilated bronchus, a tympanitic or amphoric note.

Auscultation. The respiratory murmur is bronchial, or over a dilated bronchus has a hollow sound. At the base breath-sounds are feeble, distant or absent. Râles are also heard.

The disease runs a very chronic course attended by cough, and muco-purulent, sero-purulent, and sometimes bloody expectoration, even hemorrhage; but there is no fever and not much loss of flesh. Dyspnœa occurs on ascending heights only. Dilatation of the right heart is liable to ensue, with cardiac murmurs and increased lateral dulness and increase of dyspnœa. Death is hastened by the disease, and is often brought about by an acute pneumonia.

In *pneumonokoniosis* (also known as *anthracosis*, coal miner's disease; *siderosis*, from metallic dust; *chalicosis*, from mineral dust, as in stonecutter's phthisis), there is a history of exposure to the irritating particles for a considerable period, during which time cough develops, gradually increases, and the general health fails. Emphysema simultaneously arises, causing dyspnœa. The patients wheeze, cough in paroxysms, and expectorate sputum which contains the dust particles. In anthracosis it is black. On microscopical examination, the special dust particles are often found. The symptoms of emphysema and chronic bronchitis become paramount. Tubercular infection may take place late in the disease.

Pulmonary Tuberculosis.

For convenience of diagnosis the specific inflammation of the lungs caused by the bacillus tuberculosis will be considered in this section. If a strict ætiological classification were followed it should be considered among the infectious diseases.

Clinically, we see tuberculosis in the lungs made manifest in one of the forms of acute pneumonic phthisis, acute miliary tuberculosis, and chronic ulcerative phthisis.

Definition. Tuberculosis of the lungs, pulmonary phthisis, and consumption, are names applied to an infectious and mildly contagious disease of the lungs, caused by the tubercle bacillus, appearing in an acute and chronic form, and characterized by cough, fever, sweats, more or less rapid emaciation, purulent expectoration containing elastic fibres and tubercle bacilli, and by peculiar physical signs.

Acute Pulmonary Tuberculosis, Acute Phthisis, Acute Pneumonic Phthisis, or Galloping Consumption, may be primary, or be secondary to a localized area in the lung causing rapid infection, or to tubercular pleurisy, tubercular peritonitis, or tuberculosis of some other organ. Its onset is usually marked by cough, fever with or without chills, dyspnœa, and sometimes hæmoptysis. The fever rises to 103° or 104°, and is of a continued type, or rapidly assumes a hectic type, accompanied by restlessness and exhausting night-sweats, anorexia, and rapid emaciation. Prostration of strength is extreme, but the mind is at first clear and the spirits cheerful. Cough increases, the expectoration, at first mucoid and scanty, but often tinged with blood, becomes more copious and muco-purulent. The bowels may be loose or constipated.

When death takes place without more decided pulmonary symptoms, the tuberculosis has been secondary to tuberculosis elsewhere, or death is the result of a general miliary tuberculosis.

When the acute pulmonary tuberculosis is primary the character of the disease is soon made clear by the early development of consolidation of the lungs, usually of an apex first, rapidly followed by softening and the formation of cavities. The sputum becomes muco-purulent, then purulent, is frequently streaked with blood, and pure blood is often coughed up. The sputum contains yellow elastic tissue and abundant tubercle bacilli.

The patient often presents a cachectic appearance; emaciation has been very rapid, and has reached an extreme degree; there is frequently a red flush about the cheek-bones, which, with the bright eyes, contrasts strongly with the hollow cheeks and temples, and the white wasted hands and clubbed fingers with bluish nails.

The patient's mental attitude is usually peculiarly and characteristically hopeful. He expresses himself as better each day, though occasionally subject to despondency, and is sure that if he could only gain a little strength he would soon be well.

Sometimes, especially in children, the disease is latent. The patient suffers from weariness, the cheeks flush easily, the pulse is readily disturbed, there are nocturnal fever and occasional sweatings. Emaciation proceeds very gradually, and a long time may elapse before any disease is demonstrable.

In a few cases the cerebral symptoms are so pronounced as to mask the pulmonary, and in other cases there is actual coincident involvement of the cerebral meninges.

The *physical signs* are those of consolidation, often with conjoint pleurisy. The apex is usually first invaded. There is diminished movement, increased fremitus, and dulness on percussion. At first the breathing is broncho-vesicular. It rapidly becomes bronchial. At first small moist râles are detected. Later they become large and gurgling. A pleural friction may be heard. It may first be heard above the spine of the scapula behind, above the clavicle in front, or high up in the axilla. The upper lobe of the right lung may be affected first, or the anterior portion of the middle lobe. The physical signs may be observed first in the axillary region of either side. The consolidation extends to the remainder of the lung, being preceded by physical signs indicating gradual encroachment upon the air-containing structure. The respiratory murmur is harsh, but soon becomes broncho-vesicular and then bronchial. As consolidation progresses in the middle and lower portions of the affected lung, signs of cavity or multiple cavities appear in the upper. (The whole of a lobe may be the seat of small cavities filled with muco-purulent or purulent fluid.) Cavernous breathing and pectoriloquy, or the bronchial sniff of consolidation, becomes more pronounced. The dull note of consolidation is relieved by a dull tympanitic or full tympanitic note. Now moist râles of all degrees are heard. Above they are gurgling; below, small and large moist râles. If the progress is not too rapid throughout the lung first affected, signs of invasion are found in the remaining lung, usually at a point corresponding to the primary focus in the original lung. The apex, therefore, is first invaded in most cases. Infection of the second may begin earlier than the signs in the first lung would lead one to anticipate. The rapid

invasion of one lung compels compensatory emphysema of the other. The increased movement, with harsh or puerile breathing, without change in fremitus or in pitch and tone on percussion, mask any small consolidations.

The expectoration becomes more purulent as the disease progresses, and may be blood-tinged. It is copious and possesses some fœtor. It is found to swarm with bacilli and to contain yellow elastic tissue. Hemorrhage may take place. The general symptoms become more alarming. The fever becomes of a hectic type. The patient rapidly emaciates. Cyanosis is shown in the dusky countenance and blue finger-tips. The exhaustion becomes extreme. Pallor, with flushed cheeks and an anxious countenance, is seen. The sweats are profuse. The appetite is lost. Diarrhœa may set in. Remissions may take place, even in acute cases; for a time the fever and more aggravated pulmonary symptoms are in abeyance. The typhoid state ensues in some cases. Death takes place from exhaustion and heart-clot or from meningeal tuberculosis. The duration is from two to six weeks.

Diagnosis. In the earliest stages, before the invasion of new territory is pronounced, the cases are involved in doubt. It may be confounded with pneumonia until the sputum is secured or bacilli found.

In pneumonia we have the pronounced rigor, the rapid rise of temperature, the altered pulse-respiration ratio, the hot dry skin, the sticky viscid sputum, containing the pneumococcus, the peculiar changes in the urine, the occurrence of herpes, the termination by crisis, to point to the nature of the process. The sputum is more purulent in acute pneumonic phthisis. Then cavity formation does not take place, or at least rarely. Emaciation is not marked, sweating does not occur corresponding to the repeated drenchings we see in pneumonic phthisis; anæmia is not so pronounced. In pneumonia the fever is of a continued type; in phthisis it is often intermittent or remittent. Finally, the history of exposure to infection by the disease, the primary occurrence of tuberculosis elsewhere, the secondary occurrence of tuberculosis in other organs after the lung invasion, the longer duration—aid in determining the true affection. Inoculation of animals may be resorted to in doubtful cases.

Acute Miliary Tuberculosis is attended by high fever, rapid emaciation, hurried breathing, rapid pulse, duskiness of face and extremities, more or less stupor, delirium, and the development of the typhoid state, with prostration and the occurrence of profuse sweats. Intestinal symptoms, as flatulency and distention, may be pronounced, and diarrhœa a prominent feature. *Physical signs* are negative or are those of bronchitis. There is resonance or hyper-resonance on percussion. The latter is not uncommon. The onset is abrupt or may follow a period of malaise. In some instances the tuberculous process is more advanced in some situations than in others, giving rise to special local symptoms. Thus, recently a patient was admitted to the Presbyterian Hospital with stupor and moderate delirium. He had fever, rapid pulse and breathing, and a peculiar dry, harsh skin. There was albuminuria, casts and blood in the urine, and it was thought he had uræmia. The temperature range was irregularly intermittent. The

diagnosis was established later because of the development of undoubted secondary tuberculosis in other organs. At the autopsy general tuberculosis was found, with primary tuberculous ulceration in the bladder, the ureters and renal pelves.

Diagnosis. Hurried breathing and cyanosis are distinctive features, out of all proportion to the physical signs, and, on this account, of diagnostic significance. It must be distinguished from typhoid fever, septicæmia or pyæmia, and malignant endocarditis. From the former it is distinguished by the difference in character and relations of the general and special symptoms to the period of the disease. In typhoid fever the evolution of the disease, rather than its symptoms, is characteristic. The headache of the first week, finally disappearing, is noteworthy. The special range of temperature, the onset, the fastigium, and the defervesence at definite periods in the evolution of the disease, are of diagnostic value. Cyanosis is more constant and marked in tuberculosis. The skin and capillaries have more tone in typhoid fever than in tuberculosis, at least in the first two weeks. Hyperæmia follows irritation in typhoid; pallor, with duskiness, in tuberculosis. The eruption, with its specific mode of development, belongs to typhoid fever alone. The stools, the enlarged spleen, the vascular tone are suggestive. Bacteriological examination may be of service. The occurrence of intestinal hemorrhage, pointing as it does to typhoid fever, is a welcome sign in cases in which the diagnosis is obscure. I have never seen it in tuberculosis. In typhoid fever the reflexes (knee-jerk) are never absent; in tuberculosis, if the meninges are involved, they are variable, present one day, absent the next. The diazo reaction in typhoid is of service, although it also occurs in tuberculosis. In the latter, unlike typhoid, it does not come on until later than the fifth day. It disappears at a proper time in the involution of typhoid; it continues in tuberculosis.

The distinction of tuberculosis from septicæmia or pyæmia and malignant endocarditis is often difficult. Search must be made for local areas of septic or pyæmic infection. The ears, the bones, the veins, the heart, the pelvic organs in females, the rectum, the genito-urinary tract—must be carefully examined. Hemorrhagic infarcts, or metastatic abscesses, may be found which point to the original conditions. The eye-ground may show hemorrhages. The skin and mucous membranes may exhibit minute capillary hemorrhages or infarcts. They are of the size of a pin-head, do not disappear on pressure, and are not elevated. The spleen is more likely to be enlarged in the septic affections. The respirations are not so rapid as in tuberculosis. Cyanosis is a distinctive feature of tuberculosis. The physical signs of endocarditis may be determined, and subsequently embolism or thrombosis prove the nature of the process.

Chronic Tuberculosis; Chronic Ulcerative Phthisis. Chronic tuberculosis or phthisis is much more common than acute tuberculosis, from which it is distinguished by its slow progress and by periods of remission during which the disease may be arrested temporarily or permanently.

It may begin in a variety of ways. The most common mode of origin is in an ordinary bronchitis with which pleurisy is occasionally

associated. Previous to this the patient may have been in good health, but generally the health has been impaired for some time. The bronchitis may be simple or be part of influenza, measles, whooping-cough, or some other specific disease.

The bronchitis usually proves obstinate, and by and by there is found at the apex of the lung a small area over which on percussion there is increased resistance, with slight impairment of resonance, as compared with the other side; the respiratory murmur is broncho-vesicular, sometimes jerky in rhythm, and the vocal resonance and fremitus slightly increased or unaltered. Such physical signs are met with more frequently at the right apex than at the left, and oftener in the supra-scapular fossa than anteriorly. The next most frequent seat is probably between the clavicle and second rib anteriorly.

The patient will be found to have lost strength, and usually some weight. There is often a slight evening rise of temperature, and occasionally nocturnal perspirations. The appetite is impaired, and anorexia may exist. Cough is rarely absent, especially during the night or on waking in the morning; it may, however, be so slight as apparently to have escaped the notice of the patient. When characteristic it is dry and hacking. Expectoration is scanty and mucoid, but occasionally it may be tinged with blood. It should be remembered that children and old persons sometimes do not expectorate, and that, as a rule, women are more inclined to suppress expectoration than men. No tubercle bacilli may be found in the sputum after repeated examination, but if examinations are continued they will appear sooner or later.

Instead of developing after a bronchitis, as we have just described, it may set in suddenly under the guise of a pneumonia, more frequently of the catarrhal form. The symptoms and physical signs do not differ essentially from those of pneumonia except that the expectoration is more likely to be profuse, muco-purulent and blood-streaked, and bacilli are found in it; the fever is more hectic in type, and night-sweats are common. The consolidation is found at the apex. After the patient convalesces from such an attack he continues weak, does not gain flesh readily, still has a cough with expectoration, evening fever with occasional night-sweats, and an area of consolidation usually at an apex of the lung. Over this area, in addition to the usual signs of consolidation (bronchial or feeble breathing, dulness, etc.), moist or dry subcrepitant râles are heard.

In some cases, fever, emaciation, and weakness progress for some time before pulmonary symptoms arise.

In still other cases the invasion of the disease is by sudden hæmoptysis, which is oftener copious than not. Several such hemorrhages may occur in rapid succession, or there may be only one. Moreover, its disappearance may not be followed, or least not immediately, by any further pulmonary symptoms or physical signs; more commonly, however, it is followed by a fever, cough, expectoration, and physical signs of incipient consolidation, usually at the apex.

In still other, but rarer cases, the pulmonary disease is latent, being masked by gastric or peritoneal symptoms, or by a general anæmia.

By whatever path invasion comes the physician should be on the

lookout for it, especially in a young adult predisposed by heredity or environment to tuberculosis. The recognition of the disease in its early stage requires the greatest skill, which in turn is recompensed with the highest reward, since the disease is then curable.

The further progress of a case of tuberculosis of the lungs, after consolidation has once become manifest, is very variable. It may be arrested at this point permanently, cure resulting from cicatrization. More frequently there is temporary arrest of the process; fever lessens or ceases entirely, the pulse resumes its normal rate, appetite improves, and there is a gain in flesh and strength. Cough and expectoration are more likely to persist than the other symptoms, but with the other improvement they lessen in frequency and copiousness. There are fewer râles, but the signs of consolidation are still present, though there is no further extension of the process. Often, after a cavity has been found, the disease is arrested, does not progress, or progresses very slowly.

After a longer or shorter time, as the result of re-infection from the old focus excited by acute bronchitis or by some depressing influence, the tuberculosis is re-lighted, so to speak, and runs much the same course, the lung being left more diseased and the general health worse after every such attack. Nevertheless there may be long intervals between such attacks, the patient in the meantime continuing in fair health. Thus the disease may linger or recur for years, the patient not ill enough to be confined to the house, and not well enough to stand hard work or great exposure. Slowly, by ulceration and suppuration, the lung tissue is wasted and cavities formed. Before there are large cavities at an apex the base of the same lung becomes consolidated by the production of tubercular material, and before one lung is extensively diseased the apex of the opposite lung is attacked, the process being repeated in it if the patient live long enough. Instead of re-infection from an old focus, new infection may take place, giving rise to the old train of symptoms, or setting up more acute disease. During this time the patient is liable to an attack of acute pneumonia, pleurisy, bronchitis, or general miliary tuberculosis. He is also liable to sudden death by hemorrhage. In a number of cases the intestines and peritoneum become affected, and abdominal pain and diarrhœa become superadded as symptoms.

The progress of the patient is downward. The later stages are marked by increasing cough and dyspnœa, which are very distressing and prevent sleep. Expectoration is more copious, is purulent, and is raised with increasing difficulty.

The appetite is poor and capricious, or anorexia is complete. The heart becomes more and more feeble, the fever is hectic and accompanied by exhausting night-sweats, the feet and limbs swell, and acute cramp-like pains are felt in the legs, probably caused by thrombosis of the veins

Emaciation is extreme, scarcely anything but skin and bone being left. Death occurs from perforation of an intestinal or gastric ulcer, from hemorrhage, or more commonly by exhaustion and asphyxia from œdema of the lungs.

The *physical signs* depend upon the lesions. It is often possible to detect all stages of the tubercular process, from early consolidation to large cavity, in the same patient. The signs of consolidation have been sufficiently dwelt upon. When softening begins, the percussion note continues dull and the breathing bronchial; but it is often difficult to make out the quality of the breath-sounds because they are feeble and obscured by numerous moist crackling râles and moist subcrepitant râles from disintegration of lung tissue and bronchitis. After the patient has coughed several times and expectorated, and then takes a long breath, the quality of the breathing becomes perceptible. As the lung tissue is further softened and removed by expectoration cavities are formed. These, if large enough and superficial, give a tympanitic note on percussion, and if there is a communication with a bronchus, a cracked-pot sound. The breath-sounds are hollow and cavernous, and the râles are bubbling and gurgling, or large mucous râles. The normal vocal resonance is replaced by bronchophony and pectoriloquy. Tactile fremitus may or may not be increased (see Cavities, page 263).

But if the walls of the cavity are thick from indurated tissue the percussion note will be dull and the breathing bronchial. If the tissue composing the wall is less thick and dense, percussion produces a wooden sort of resonance. If much normal lung tissue intervenes, the percussion note will be clear.

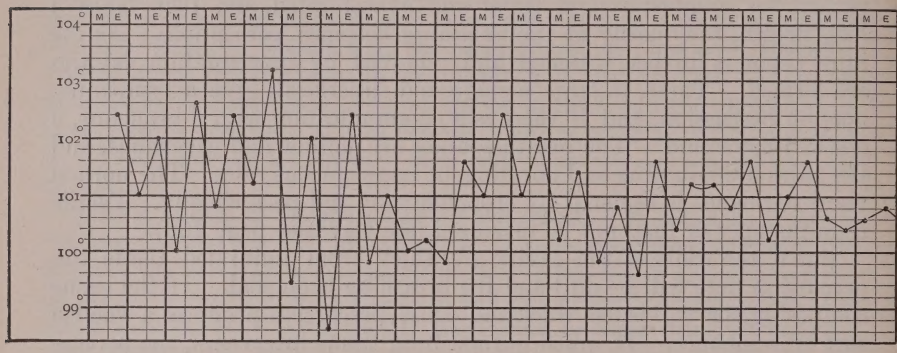
As tuberculosis of the lungs progresses the clavicles and ribs become more and more prominent from the loss of fat, and local flattening of the chest with impaired expansion marks the seat of the disease.

The Diagnostic Features. The striking phenomena of tuberculosis which are considered in the diagnosis are emaciation, anæmia, fever, cough, dyspnœa, chest pain, hemorrhage, the expectoration, and the objective symptoms. Of less diagnostic value, but important as collateral data, are the aspect, the occurrence of vomiting and diarrhœa, and of symptoms of secondary tuberculosis in other organs. Reliance can be placed, to a certain degree, upon such associated circumstances as age and occupation, in the formation of the diagnosis.

Emaciation. This is always seen, even in acute forms of tuberculosis. It is rapid in the acute, slow and progressive in the chronic forms. In the latter the flesh may be restored for a time. It must not be confounded with muscular atrophy, and the emaciation of carcinoma, diabetes, anorexia nervosa, and other exhausting diseases. *Anæmia* is always pronounced. It may be associated with leucocytosis. The reduction of red cells and diminution of hæmoglobin are marked. *Fever.* This symptom is always present. The temperature should be taken every two hours for a time, to determine accurately the degree and course. It may be intermitting, remitting, or continuous. It may be intermitting in some acute forms, the morning fall reaching or going below normal. The difference between morning and evening temperature may not be more than a degree. In the acute forms it is high and continuous, and soon may be attended by the typhoid state. In the more chronic cases it may be intermittent at first, then continuous, and finally intermittent again. In the later stages the intermitting fever is due to a mixed infection, or sapræmia, from the purulent contents (staphylo-

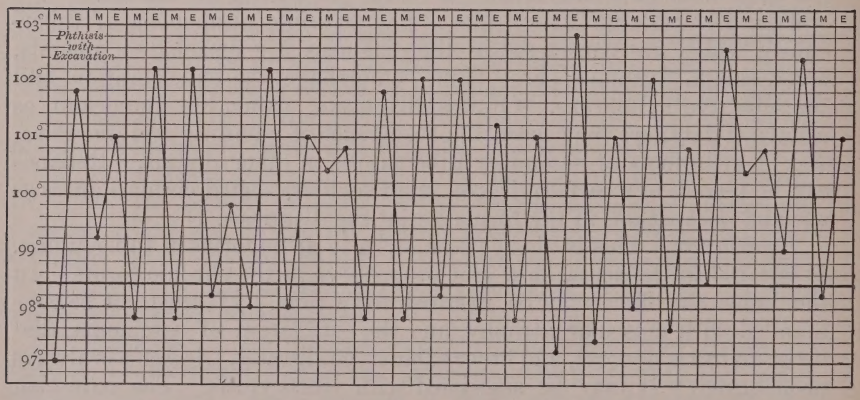
coccus and streptococcus infection) of the lung cavities[1] (see Fig. 57 and Fig. 58). The intermittent fever of the early stages has frequently been mistaken for malaria (see Fever). The occurrence of fever in a patient who has been losing flesh, and is otherwise in poor health, excludes cancer and diabetes and other afebrile causes, and points strongly to tuberculosis. It must not be forgotten that in chronic tuberculosis in

FIG. 57.



Continued fever of tuberculosis.

FIG. 58.


103°
Phthisis with Excavation
102°
101°
100°
99°
98°
97°
M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E M E

Intermitting fever of tuberculosis.

the aged the temperature may not rise above 100°; often, indeed, it is subnormal.

We must consider, therefore, that fever, the cause of which is not obvious, may be due to tuberculosis; that, if not controlled by allaying probable causal conditions, as gastro-intestinal catarrh or infectious disorders,

[1] Leyden has recently pointed out that intermitting fever is part of the tuberculous process and not a strepto- or staphylococcus infection as formerly held, because pus micro-organisms are not found in the purulent contents of cavities, and because in other forms of tuberculosis, as empyema or joint disease, they are notably absent, and yet such form of fever exists.—*Deutsche medicin. Wochenschrift*, Sept. 14, 1894.

as malaria, or relieving suppurations, it is more probably of tuberculous origin.

Sweats. Sweating, which is frequent, may be the first symptom complained of by the patient. It may occur with the tripod of symptoms of the intermitting febrile range—chill, fever, and sweat. It would be likely to occur at night under these circumstances. It may occur at any time, however. "Night-sweats" are alarming to the mind of the laity, and truly of diagnostic significance. The perspiration awakens the patient at night because it is so profuse. It may be moderate only, not rousing the patient until morning. It may be general or local. Local sweats are confined to the head and neck. *Anœmia.* This quite rapidly becomes marked. It is recognized by the color of the surface and by an examination of the blood. When collateral inflammation is present, leucocytosis is seen. *Cough.* Cough is one of the earliest symptoms. It may be the single symptom for some time. It is often dry and hacking at first. Such dry cough may continue for a long time. Later, it is accompanied with mucoid and then muco-purulent sputa, which contain the characteristic elements (see Sputum). *Dyspnœa* is almost always present. The degree varies with the association of fever. When the latter is present, dyspnœa is more pronounced. It is more pronounced in acute cases. In miliary tuberculosis the frequency of respirations that attends the dyspnœa is out of all proportion to the physical signs. In this form, cyanosis is more marked. In chronic localized phthisis, the dyspnœa may only occur on exertion, after eating, or upon excitement. The bloodless lips may have a constant bluish hue. The fingers are dusky and become "clubbed." In the later stages the dyspnœa is constant and in proportion to the extent of involvement of the lungs and the degree of fever. Although of diagnostic significance only with other symptoms, it is most distressing, and is the cause of constant demand for relief.

Chest Pain. This is due to localized pleurisy or to myalgia. The latter may be seated in muscles strained by coughing. Pleuritic pains may occur in any situation, and vary in position from time to time. They may be due to extensive inflammation or to tuberculous pleurisy. Constantly recurring and unilateral chest pains, with or without signs of pleurisy, with cough and emaciation, are significant of the disorder under consideration (see Pain). *Hemorrhage.* This symptom is alarming, and, in the large majority of cases, due to pulmonary tuberculosis. It may mark the onset of the acute disease, and continue irregularly throughout its course or recur several times before the advent of more common symptoms of the chronic form. It may occur at intervals of a few months, or a year, before emaciation, cough, and characteristic expectoration set in, or before bacilli are found in the sputum. Each attack is attended by fever, usually, and followed by anæmia and prostration. If hemorrhage of the lungs (see Symptoms) occurs in a young adult without cause (as aneurism or cardiac disease, etc.), it must be looked upon with suspicion. The likelihood of tuberculosis is increased if the bleeding occurs in a patient of tuberculous aspect, in whom a family history of tuberculosis is found, and who has

been exposed to infection. In the aged it may occur from a localized area of disease.

Hemorrhage is also common in the late stages of tuberculosis. It is not at this period of diagnostic value as to the primary cause. It is usually due to the erosion of an artery in a cavity.

Hemorrhage also occurs in tuberculosis in the quiescent period. The progress of the disease is arrested. The discharge of blood is accompanied by the expectoration of pulmonoliths, calculi formed by the degeneration of caseous areas.

The Sputum (*q. v.*). The diagnosis is absolute when tubercle bacilli are found in the expectoration. Nummular sputa are more common in phthisical excavation. The sputum is discharged in tough coin-shaped masses which sink when expectorated into a vessel containing water. Fragments of lung tissue (yellow elastic) point to tuberculosis, but are possible under other circumstances.

The Physical Signs. The objective signs point to invasion of air-containing structure by solid material, with collapse of lobules, to consolidation and to cavity formation, and to the secondary occurrence of pleurisy. In the chronic cases, contraction, lessened movement, dulness and increased resistance from thickened pleura may override the signs of consolidation. No one physical sign is of diagnostic significance. The combination of signs, and the orderly procession by which they advance as the physical conditions progress, are the most diagnostic. Local contraction (flattening) and impaired movement at an apex, with suppressed breath-sounds and prolonged expiration, with impaired resonance, are the earliest signs of tuberculosis.

The *aspect* of the patient is always suggestive, and is an aid to the recognition of the condition. The tuberculous or phthisical chest, the long neck and arms, the pale face, the occasional hectic flush, the clubbed fingers, the emaciation, of the many subjects we see in our infirmaries, fix in our minds a composite picture the recognition of which in individual cases goes far to diagnosticate the insidious disease. *Vomiting* (see Gastro-intestinal Disease) is a symptom which is often present in the early stages of tuberculosis of the lungs, and frequently masks the true condition. The vomiting may lead to the belief that a local gastric catarrh or diarrhœa is to blame for the general symptoms. The occurrence of fever with the gastric symptoms should lead to an examination of the lungs.

The occurrence of diarrhœa and symptoms of tuberculosis in other organs may thoroughly establish a diagnosis in tuberculosis of the lungs with otherwise obscure pulmonary symptoms. The intestinal discharges may contain tubercle bacilli, or the latter may be found in the urine in joint suppuration or glandular enlargement.

In addition to the above we might carefully associate circumstances. The age is inquired for, adolescence and early adult life being the common periods in which pulmonary tuberculosis develops. The occupation,[1] the history of exposure to the disease, the history of predispo-

[1] Several undoubted instances are recorded in which hospital residents and young physicians working in laboratories in which tuberculosis is studied, or constantly examining sputum, have been infected in the course of their studies.

sition to tuberculosis in the family, the history of previous, now arrested, tuberculosis, as in joint disease, or glandular tuberculosis (scrofula), are data deserving special consideration, which are corroborative evidence of the presence of the disease.

The Diagnosis is Established by Finding Tubercle Bacilli in the Sputum. Their absence, careful search having been made, is against the tuberculous origin of the disease.

In subsequent chapters the differential diagnosis of tuberculosis and other diseases will be pointed out. It must not be forgotten that the disease may set in and be the terminal affection in many diseases. Thus, in diabetes, in insanity, in chronic cerebral or spinal disease, and in other affections, tuberculosis may develop insidiously, and finally cause death.

It must be distinguished from chronic gastric disorders, and particularly anorexia nervosa. It must not be confounded with malaria. It must be distinguished from simple anæmia, the cause of which may be recognized with difficulty. It must be distinguished from chronic bronchitis with bronchiectasis, from pulmonary gangrene and carcinoma. Finally, it must not be mistaken for cancer of the œsophagus and aneurism of the aorta, two divergent conditions which may have pulmonary symptoms simulating phthisis.

Gangrene of the Lung.

Gangrene is a rare disease of the lung, and, like abscess, always secondary. It may be produced by any cause which so obstructs the circulation that a portion of lung dies in bulk. The gangrene may be circumscribed or diffused; it results most frequently from pneumonia, but may be due to injury, to a general septic condition, or to embolism. It is relatively frequently met with in the insane, possibly owing to particles of food which have found their way into the lung. Aspiration broncho-pneumonia, bronchiectatic and tuberculous cavities, sometimes lead to gangrene. Gangrene in the lung, as elsewhere, occurs in diabetes.

Symptoms. When it occurs in the insane or is of embolic origin it may remain latent, and in septicæmia it may be overlooked on account of the general symptoms. In well-marked cases, however, the symptoms are characteristic. Symptoms and physical signs of pulmonary disease precede the specific symptoms of gangrene. With the onset of a moderate fever hæmoptysis may occur at once or be preceded by the expectoration of a brownish, purulent sputa having a most intense and persistent gangrenous odor. It contains fragments of lung tissue, altered blood, and putrid débris. (See Sputum.) It separates into the three characteristic layers in a conical glass. The fœtor of the breath and sputum is diagnostic.

The disease usually occupies the lower or middle lobe of the lung. The *physical signs* are those of cavity. The disease could with difficulty be distinguished from abscess except for the characteristic sputum, though in gangrene there is greater tendency to a general septic condition, with profuse sweats and collapse.

Abscess of the Lung.

Abscess of the lung may originate in causes outside the lung, or in causes within the lung. To the former class belong those produced by suppurating bronchial glands, abscess of the mediastinum opening into the lung, cancer of the œsophagus with ulceration, and abscess of the liver, suppurating hydatid cyst, or sub-diaphragmatic abscess in general, bursting into the lung. Intra-pulmonary causes are tubercle, septic emboli, in which case the abscesses are multiple and sub-pleural, and pneumonia. In the aspiration form of lobular pneumonia abscesses occur. Rarer causes are the presence of tumors and obstruction of the bronchi.

Abscess of the lung is therefore always secondary. Its diagnosis depends upon the demonstration of a cavity taken in connection with the history pointing to a cause. The sputa are copious, purulent, often odorless, sometimes offensive but always without the fœtor of gangrene. They contain elastic fibre, but no bacilli except in tuberculous cases (see Sputum). In embolic abscess the signs of pleural friction can only be detected at times.

Collapse of the Lung.

Collapse of the lung is a condition produced by exhaustion of air from the air-vesicles. It may affect alveoli here and there, or a large section of the lung. Formerly such collapse was invariably looked upon as pneumonia until Legendre and Bailly proved by forcible inflation that the air-vesicles had simply collapsed from absence of air. Collapse occurs most frequently in the course of bronchitis and in cases with feeble respiratory power. The bronchial twigs supplying certain air-vesicles, or tubes supplying sections of lung, become occluded to such a degree that no air can enter. The air already contained in the vesicles then becomes exhausted gradually until the vesicles are completely airless. The vesicles or sections of lung involved then return to their fœtal condition. When the collapse is congenital the term *atelectasis* is preferable. Anything which induces great muscular weakness predisposes to collapse of the lung; hence in the aged and feeble, in wasting diseases, and in low febrile diseases of long standing, collapse is very liable to occur. But bronchitis is the most frequent and direct cause. The secretions which are poured out, and the swelling of the mucous membrane, occlude the tubes, and if the patient have not strength enough to expel the secretions, and, by forced inspiration expand the collapsing vesicles, collapse ensues.

Diagnosis. The diagnosis of the condition in life is difficult. The area of collapse, being airless, is, of course, dull on percussion. The respiratory murmur is more likely to be faint or absent than to be increased in intensity or approach the bronchial. Nevertheless there is sometimes heard a faint broncho-vesicular expiration.

When œdema is superadded to collapse, moist crepitant râles are heard, difficult if not impossible to distinguish from those of pneumonia. Respiration is embarrassed, and is accompanied by sucking in of the

lower part of the chest in inspiration. Sometimes the plug of mucus which occludes the tubes becomes dislodged while the physician is auscultating, and then the respiratory murmur will be heard accompanied by a succession of crepitant râles, which disappear after a few inspirations. The dull areas, as a rule, are less persistent than those of pneumonia; thus it may be found at successive examinations that one area has cleared up and another has become dull. Stress is laid by some writers upon the signs of emphysema surrounding collapsed areas. But this does not give assistance in the cases in which most help is required —cases in which there is diffuse bronchitis with more or less œdema.

Subjective symptoms are those of dyspnœa and deficient oxygenation of the blood. If these are developed suddenly, and are accompanied with the appearance of dull areas in the lung without bronchial breathing, the diagnosis is tolerably certain. But when scattered lobules only are involved the physical signs of collapse are absent, and its existence must be a matter of inference.

From *lobar pneumonia* the diagnosis is generally easily made by the difference in the physical signs, and by the absence in pulmonary collapse of inflammatory symptoms, the lower temperature, and the difference in onset.

The diagnosis from *broncho-pneumonia*, or *catarrhal pneumonia*, is beset with greater difficulties. But here also the lower temperature and the fact that the physical signs and the location of the dull areas are subject to rapid changes, is of aid in diagnosis.

Cancer and Other New Growths of the Lung.

The new growths may be primary or secondary. The latter are most common. Of primary cancer, the epithelioma is most common; encephaloid and scirrhus come next. Sarcoma is sometimes primary. Secondary new growths succeed disease in the abdominal organs, the genito-urinary tract, the bones, the breast, and the eye.

Symptoms. The general symptoms of malignant growths accompany the thoracic symptoms. Chest pain, dyspnœa, cough, and a peculiar expectoration belong to the latter. The pain is due to associate pleurisy; the dyspnœa is paroxysmal. (See dyspnœa from pressure on bronchi.) The expectoration is dark, like prune-juice. Signs of intrathoracic pressure are seen. The external thoracic veins are enlarged. The face and arms may be cyanosed, or one arm only affected. The heart may be dislocated, the trachea changed in its course; compression of trachea and bronchus causes dyspnœa.

Physical Signs. In primary cancer the affection is unilateral; in secondary forms, bilateral. The physical signs are those of pleural effusion or of local consolidation. The consolidation may be massive and not partake of the shape of a lobe. Often signs of effusion and consolidation are combined (enlargement, immobility, absent fremitus, but bronchial breathing). In the secondary forms the disease is bilateral. The signs are mixed. They indicate lessened air in the lung structure. Care must be taken not to overlook the pleural effusion which accompanies the process, the removal of which gives temporary relief. In

both forms external lymphatic glands, particularly the cervical, may be enlarged.

Diagnosis. The diagnosis is based upon—1, the age (after forty); 2, the occurrence of emaciation; 3, the duration of the disease, often rapid, rarely beyond eight months; 4, the presence of primary disease elsewhere; 5, the occurrence of moderate fever; 6, the signs of intrathoracic pressure; 7, the involvement of lymphatic glands; 8, the occurrence of irregular areas of consolidation and of pleural effusion, alone or combined; 9, the characteristic expectoration; 10, dyspnœa due to pressure on the bronchus or trachea; 11, the absence of bacilli from the sputum.

An effusion often can be recognized only after puncture. Hæmothorax is not necessarily present.

Hydatid Disease of the Lungs.

The lungs are affected in about 11 per cent. of the cases of hydatid disease. The symptoms, according to Wilson Fox, consist of dyspnœa, pain in the chest, cough, occasional hæmoptysis, and sometimes the expectoration of hydatids, the sputa being otherwise bronchitic, or presenting the characteristics of pneumonia or gangrene, when these complications are present. Gradually weakness increases, sometimes with pyrexia, which, when combined with emaciation, may impart to the case a considerable resemblance to phthisis; pressure symptoms occasionally occur, and the physical signs are either of consolidation of the lung or of pleural effusion, together with certain peculiarities depending on the size and site of the tumor. Graham states that they are more frequent in the right lung and more common at the base, causing marked bulging of the thoracic wall. The physical signs are those of pleural effusion with localization of the fluid to a definite area, and hence not related to the shape of the pleural cavity. The breathing may be tubular; there is condensed lung between the hydatid and the thoracic wall. Cough, dyspnœa, anæmia, with emaciation and clubbing of fingers, lead to the diagnosis of phthisis. Hæmoptysis occurs in many cases. The temperature is normal—an important point in diagnosis. If the cyst ruptures the sputum is diagnostic. Complications mark the diagnosis often. It must be distinguished from pleurisy, localized empyema, pulmonary abscess, phthisis, and mediastinal tumors.

Diseases of the Pleura.

The large lymph structures which cover the lung and line the inside of the thorax are often the seat of disease. It is usually of an inflammatory nature. Hence, pleurisy, or pleuritis, is the most common affection of the pleura. It may be, as to distribution, bilateral or unilateral; as to extent, local or general; as to the nature of the inflammation, plastic, serous or purulent. The inflammation may be acute or chronic. It is rarely primary. It arises in the course of general diseases, or is the result of the extension of inflammation, chiefly of an infectious nature, from neighboring structures.

1. Disease of the ribs or vertebræ, diseases of the mediastinum, of the aorta, œsophagus, and especially of the lung, give rise to various forms of pleurisy, depending upon the nature of the primary affection.

2. Disease below the diaphragm. Abscess of the liver; perforative inflammation of other viscera adjacent to the diaphragm; abscess of the spleen or pancreas; pus in the pelvis or about the appendix, may give rise to purulent pleurisy by burrowing of the pus or infection through the lymph channels.

3. Disease of the lungs. In the large majority of cases pleurisy in some form occurs in the course of pulmonary disease. In all surface inflammations of the lungs there is associate pleurisy. It is seen in pneumonia, in tuberculosis, in gangrene, and in abscess.

Pleurisy may be simple or purulent. Empyema is always due to infection from the exterior, as the ribs; from the lungs (pneumonia); suppuration below the diaphragm; or to general infective processes, as septicæmia, pyæmia, and tuberculosis.

The general diseases in the course of which pleuritis arises are usually infective or of such nature as to cause irritative products to circulate in the blood. Of the former, the most common is tuberculosis; the next most common are septicæmia and scarlatina; while to the latter class belong Bright's disease, gout, diabetes, rheumatism, and scurvy.

Purulent pleurisy is more common in children than in adults; in males than in females; and more common in tuberculous pleurisy and pyæmia than in rheumatism and Bright's disease.

Acute Pleurisy.

Acute pleurisy may be primary, or may be secondary to disease of the lung, or be part of a general infection. Three stages in the morbid process usually occur, although it may be arrested in the first stage.

Symptoms of the First Stage. *Dry Pleurisy.* The *onset* of the disease is usually abrupt, and is marked by *fever*, which may or may not be preceded by chill, and is followed by *pain* in the side, *dyspnœa*, and *cough*. The pain is sharp, stabbing, or tearing in character, and is usually, but not always, referred to the seat of pleurisy. This is most frequently on a level with the nipple, or a little below this, and oftener anteriorly or in the axilla than posteriorly. The pain is caused by the rubbing together of the inflamed surfaces of the pleura, and hence is excited by respiration and cough. For this reason the patient is inclined to restrict the motion of the affected side as much as possible; he does this by leaning over toward that side and by pressing his elbow in against the chest wall. Pain is usually the first symptom noticed by the patient. The cough is dry and painful. Fever is moderate.

The *physical signs* in primary cases are a friction sound heard on inspiration and expiration. This friction sound may be a nest of fine, dry, crepitant râles, which are very superficial, and appear to be just under the ear; or a coarse rubbing sound, heard over a larger surface, and resembling a bronchial rhonchus, from which it can be distinguished by its persisting after the patient has coughed. The lungs themselves present nothing abnormal.

If the inflamed surfaces become glued together by plastic lymph, recovery usually occurs very soon, though pain often persists for a long time in lessened degree, and the pleurisy is liable to be re-lighted.

Symptoms of Second Stage, or Stage of Effusion. If *effusion* takes place the two layers of the pleura become separated; hence pain and friction sound cease, and physical exploration shows that a collection of fluid intervenes between the chest wall and the lung. The *physical signs* of this stage are (1) enlargement of the affected side, increase in semi-circumference, with fulness of interspaces; (2) diminution of movement; (3) absence of vocal fremitus and resonance; (4) dulness or flatness (deadness) on percussion, with great increase in the resistance to the pleximeter finger; (5) absent or greatly diminished respiratory murmur; (6) displacement of organs.

The dead percussion note being caused by fluid, it follows that the *upper level* of it will change with the position of the patient if the fluid is free. If the upper level is at the third interspace when the patient is sitting up, it will fall to the fourth or lower when he is lying down. This change of level cannot be appreciated when the effusion is very large. Moreover, above the line of dulness the percussion note is *hyper-resonant* or *tympanitic*—Skoda's resonance. Toward the spine on the affected side there may be partial resonance and bronchial breathing, because here the lung is compressed against the vertebræ. In large effusions the tympanitic resonance in the second interspace does not change when the mouth is opened. "Williams' tracheal tone" can often be elicited in large effusions. The upper limit of dulness in large pleural effusions is higher at the spine and slopes downward, and is lowest in front. In moderate effusions the line of dulness is lowest near the spinal column, rises in the middle of the scapula and slopes downward, assuming the shape of the letter S as it passes toward the front (Garland). The patient should take deep breaths before the percussion is performed. At the left base in front the semilunar space is removed, dulness continuing to the margin of the ribs.

Below the upper level of the effusion posteriorly the voice frequently has a metallic quality resembling the bleating of a goat—*ægophony*. It occurs usually when the effusion is moderate, and may be heard only over a limited area. It is commonly heard at or above the angle of the scapula.

While the respiratory murmur is, as a rule, absent, breath-sounds may be heard, and are then usually bronchial. In such cases there may or may not be adhesions. Bronchial breathing may be present along the spine in small effusions, and in large effusions in the interscapular region. Bronchial breathing, tubular in character, is said to be almost constant in children. It may also occur when pneumonia coexists.

At the level of the fluid a friction sound may persist. Above the level of fluid anteriorly the breath-sound may be bronchial or bronchovesicular, associated sometimes with fine râles, due to compression and slight œdema.

Displacement of Organs. If the effusion is on the left side the mediastinum and heart become displaced to the right, and the apex beat may

be found in the epigastrium, or even to the right of this. At the same time the semilunar space (Traube's line) is lower than usual or entirely effaced. If the effusion is on the right side, the diaphragm, and with it the liver, is depressed, and the mediastinal contents moved to the left.

The *subjective* symptoms during this stage are slight or moderate fever, sometimes intermittent in character, with recurring chills; considerable dyspnœa, occasionally amounting to orthopnœa when the effusion is very extensive; and dry cough, which adds greatly to the dyspnœa. There is frequently some evidence of defective oxygenation of the blood; when this amounts to cyanosis, the condition is one of great danger. The urine presents changes in amount. In advancing effusion, the amount lessens very much; it increases in amount with the decline of the fluid. Pleurisy may be complicated with bronchitis, pneumonia, and pericarditis.

Empyema. The above-mentioned physical signs apply chiefly to serous effusions. They are also present in effusions of pus. In addition, other physical phenomena and different general symptoms distinguish the two kinds of effusions, although it must be confessed that aspiration must often be resorted to.

The *physical signs of empyema* are the same as those of other effusions within the pleura. In addition, especially in children, local *œdema* of the chest-wall may be found. Another sign was pointed out by Bacelli, and is held by others to be of diagnostic significance. In purulent effusions the fremitus produced by the *whispering* voice is not transmitted to the hand laid over the effusion, whereas in serous effusions such vibrations are transmitted. In empyema a local area may become more prominent and the surface assume an inflammatory appearance. It is an indication of discharge of the abscess through the chest wall. It is usually found in the fifth interspace in front, or below the angle of the scapula behind. (For a microscopical and chemical description of the "Effusions within the Pleural Sac," and of the morphological elements of the purulent effusions, see Chapter V.)

The *general symptoms* are more marked in empyema than in simple serous effusion. The temperature is higher from the onset. It soon becomes intermittent or remittent. Chills or chilliness may attend the beginning of each febrile paroxysm, and sweats occur with the daily fall of temperature or at irregular periods during the twenty-four hours. The heart's action is more rapid and the pulse more feeble, and it soon becomes dicrotic. Examination of the *urine* may aid in the distinction of the two forms of the effusion. *Peptonuria* occurs in purulent pleurisy. It must be remembered that peptonuria occurs in suppuration from other causes. Thus, in phthisis with suppuration of a cavity pleural effusion may develop. The peptonuria that attends the primary process must not be mistaken for that which occurs in empyema. *Indican* is also present in excess in the urine in suppurations. Before a decisive conclusion is arrived at two or more examinations of the urine should be made. Examination of the *blood* may aid in arriving at a conclusion. In purulent effusion there is usually a leucocytosis.

Notwithstanding the positive physical signs of effusion the character of the effusion may not be recognized until perforation into the bronchus

has taken place. The peculiar character of the expectoration that attends this accident is described in the section on Sputum.

Hydrothorax. This is an accumulation as the result of a transudation. (For character of the fluid, see Chapter V.) It occurs in the course of diseases which produce anasarca, as failing organic heart disease, chronic Bright's disease, and debilitating diseases, as scurvy. Locally it may attend carcinoma of the pleura or obstructive disease of vessels within the mediastinum.

The physical signs of hydrothorax are those of effusion in acute pleurisy. The general symptoms belong to the primary disorder. Dyspnœa may develop gradually and even amount to orthopnœa. It is distinguished from inflammatory effusions by the character of the fluid, by the absence of the general symptoms of inflammation, by its insidious development, and by its bilateral distribution.

Hæmothorax. The transudation of blood into the cavity of the pleura occurs rarely from the rupture of an aneurism into the sac. The fluid is then pure blood. Serous effusions in which a large amount of blood is found point to primary carcinoma of the pleura, or to tuberculous disease. Both specific processes of this serous membrane may occur, however, without the transudation of sero-bloody fluid.

Thickened Pleura. Chronic inflammation, with thickening of the pleura from excessive development of connective tissue, occurs in tuberculosis and in cases of combined pleuritis and peritonitis. The physical signs are pronounced and are those of effusion without enlargement of the chest. The thickening of the pleura is usually more marked at the base. On inspection there is marked contraction and diminution in movement of the affected side. The fremitus is absent. There is dulness on percussion, or even flatness. The breath-sounds are distant or are absent entirely. Along the vertebræ, especially opposite the angle of the scapula, bronchial breathing may be heard. The subjective symptoms of cough and dyspnœa are present. The degree of cough depends upon the condition of the parenchyma of the lung. If there is bronchitis or tuberculosis, the cough is excessive. The amount of dyspnœa depends upon the degree of compression of the lung by the thickened pleura.

Tuberculous Pleurisy. The affection may be acute or chronic. It may occur primarily, be a part of general tuberculous infection, or occur secondarily to disease of the lungs. It may give rise to all forms of the inflammatory process. First, dry pleurisy; second, pleurisy with effusion; third, pleurisy with great thickening, may be found. The distinction between tuberculous pleurisy and pleurisy due to other causes can often not be positively determined. If it is associated with tuberculosis in other organs, or the patient is of tuberculous habit and exposed to infection; or, if there has been a history of previous tuberculosis, the pleuritic infection is probably of tuberculous origin. If the affection is bilateral and associated with peritoneal inflammation, and at the same time no other cause exists for serous membrane inflammation, the probability of its tuberculous origin is very strong.

Pulsating Pleural Effusion. Wilson has presented the most recent studies of this rare affection. The effusion within the pleura pul-

sates synchronously with the ventricular systole; the pulsation is detected usually by inspection and palpation. In some instances its presence is only determined by palpation. It may be confined to two or three interspaces or occupy the anterior aspect of the thorax and the axillary region on the left side. Rarely the pulsation is behind. It is usually situated on the left side. The original effusion is purulent in the large majority of cases. The physical signs and general symptoms of empyema are present. Nevertheless the disease simulates aneurism of the aorta. The latter affection, however, is accompanied by vascular symptoms and physical signs discovered in the course of the aorta. Pulsating empyema is distant from the aorta.

Diaphragmatic Pleurisy. In diaphragmatic pleurisy there is intense pain in the epigastrium. Gueneau de Mussy[1] regards a pain along the tenth rib, extending from the anterior extremity to the sternum and xiphoid cartilage, as pathognomonic. Other symptoms are nausea, vomiting, and hiccough. The dyspnœa often amounts to orthopnœa, or the patient sits stooping forward. The anxiety of the patient is very great. The fever is usually higher than in ordinary pleurisy, and there may be delirium. Effusion may lessen the pain. Peritonitis may occur at the same time, or be secondary to the pleurisy.

Diagnostic Features. The special features of diagnostic importance that are observed in the course of pleurisy are the pain, the dyspnœa, the cough, the fever, the physical signs of effusion within the pleura, and the results of exploratory puncture. *Pain:* The pain is short, sharp, lancinating, and from its character and location is usually readily recognized. It must be distinguished from the pain due to pleurodynia and intercostal neuralgia. The pain of pleurisy is associated with cough and is increased by breathing. It causes diminution of movement of the affected side. The patient is compelled to sit up in bed or lie on the side which is the seat of pain. *Cough:* In the first stage the cough is short, suppressed, dry and painful. It is constant. In the second stage it changes in character. There is no pain, there is no expectoration. It is frequent and irritating and of a peculiar sound which is difficult to describe, and yet, when once heard, is most suggestive in subsequent cases. It is short and lacks resonant quality, as if the fluid in the chest stopped the sound waves. *Dyspnœa* in the first stage is due to pain, in the second stage to the large effusion which encroaches upon the normal air-space. It is not diagnostic. The *physical signs* of pleural effusion have been frequently reiterated. The most decisive are diminution or absence of movement, enlargement of the affected side, absence of fremitus, flatness on percussion, fulness of intercostal spaces, and the displacement of organs. The latter is of the greatest diagnostic importance in the distinction between consolidation and effusions. The results of *exploratory puncture* lead to decisive conclusions usually, although it must not be forgotten that effusions may be loculated and by accident not secured by the aspirating needle. Or the enormously thickened pleura may intervene between the exudation and the surface of the chest, and prevent withdrawal of the

[1] Arch. Gén. de Med., 1853, vol. xi. Quoted by Fox.

fluid. Finally, effusions may complicate inflammatory processes, as pneumonia, tuberculosis, or abscess of the lung. To secure fluid for diagnosis by aspiration, therefore, does not necessarily exclude these conditions, and hence, before the process is decided to be within the pleura alone, the sputum and other conditions must be taken into consideration.

Differential Diagnosis. *Acute plastic pleurisy* is diagnosticated from *acute pneumonia* by the friction sound and the maintenance of the clear percussion note and normal respiratory murmur, with unaltered vocal resonance and fremitus. When *effusion* takes place the chest is enlarged and immobile, especially on the affected side, the interspaces are filled out and the diaphragm is depressed; these changes do not occur in pneumonia. Moreover, the percussion note in pleural effusion is flat, with greatly increased resistance; the shape of the upper line of dulness is diagnostic; the respiratory murmur is feeble and distant, or entirely absent, except along the spine, where the compressed lung yields bronchial breathing, and also above the line of effusion, where the lung yields exaggerated breathing. In pneumonia, on the other hand, the percussion note is dull, without greatly increased resistance, and the breath-sounds are bronchial. In addition, in pleurisy, the vocal resonance and fremitus are usually almost if not quite absent, and posteriorly at the level of the effusion, ægophony may be detected. In pneumonia, on the contrary, vocal resonance and fremitus are increased in intensity. In pleurisy with effusion the movable organs are dislocated and Traube's line is obliterated.

Finally, the fever of pneumonia is much higher and more continuous than that of pleurisy, the respirations more frequent, the cough looser and in typical cases followed by rusty sputa. A crucial test is *aspiration* with a hypodermic needle; in pleural effusion, serum is withdrawn; in pneumonia, a few drops of thick blood.

In *pleurodynia* there is also severe pain in one side. But the pain is more continuous than that of pleurisy, and consists of a constant aching or a burning sensation. It is made worse by twisting or turning, as well as by breathing. The side is also tender to the touch. The pain is not so sharply localized as that of pleurisy, and may leave one side and affect the other. It is unaccompanied by fever or friction sound, and is frequently found in rheumatic subjects.

In *intercostal neuralgia* there is the same absence of fever and friction sound. The pain, however, is sharply localized as in pleurisy, but is of the darting neuralgic character, and is associated with tenderness at the points of exit of the intercostal nerves. It is most common in women, especially if they have uterine disturbances. It is more frequent on the left side, and just beneath the mammary gland.

Chronic Pleurisy.

Chronic dry, or plastic, pleurisy is the result of an acute attack, or develops insidiously if tuberculous. It causes great deformity of the chest from contraction and compensatory emphysema of the healthy lung. The heart is dislocated or cannot be found on physical examina-

tion, because it is overlapped by lung or is drawn behind the sternum. There is considerable spinal curvature, dislocation of the scapula, deformity of the shoulder, and indrawing and overlapping of the ribs at the base of the chest.

Chronic pleurisy with effusion results from an acute attack of pleurisy, in which the fluid remains unabsorbed, or from subsequent attacks. The physical signs are the same as in acute effusion. So far as subjective symptoms go it may remain latent; patients so affected not infrequently go about their work with comparatively little dyspnœa. There may be an evening rise of temperature and acceleration of the pulse. Chronic effusions are more likely to be purulent in children than in adults. When empyema results, the fever becomes hectic; there are chills and sweats, pyæmia develops, and death is liable to occur from some intercurrent suppuration, as cerebral abscess.

After *chronic effusion* the chest is rarely restored to its original shape even if the effusion be absorbed finally. The affected side becomes motionless and retracted. In process of time the spine may be bent. The opposite lung becomes hypertrophied. The patient is usually in precarious health, liable to acute attacks of pain in the affected side, and liable also to be carried off by phthisis or some intercurrent affection. Rarely the patient may maintain good health, and even cure, with restoration of the retracted side to, or almost to, normal dimensions, is possible, especially in children.

Pneumothorax.

Pneumothorax consists in an accumulation of air in the pleural cavity, accompanied or followed by an outpouring of fluid, which may be serous or purulent, constituting respectively *hydro-pneumothorax* and *pyo-pneumothorax.*

Pneumothorax may originate: 1. From causes external to the chest, by perforation of the chest wall and pleura. 2. From perforation of the lungs, bronchi, or œsophagus. 3. Gases developed from an existing effusion.

The most frequent cause is tubercular disease of the lung, and next an empyema; out of 121 cases collected by Saussier, 81 were due to phthisis and 29 to empyema. It may occur very early in tubercle of the lung, and even be the first symptom of tubercular disease (see cases referred to by Fox and recorded by Louis and Chomel). The left side is affected not quite twice as often as the right; the disease is usually unilateral. The onset of the condition is usually sudden. During a paroxysm of coughing or vomiting, or without immediate cause, there is an escape of air into the pleura, and in the majority of cases the patient at once complains of acute pain in the chest and excessive dyspnœa with great dread of impending suffocation. The patient often sinks into collapse from shock, but sudden death is rare. If the escape of air into the pleura is gradual there will be less pain and dyspnœa. The chest is distended, especially on the affected side; the percussion note is a bell-like tympany, except when the distention is excessive and the air contained is under great tension, when the note is proportion-

ately duller and higher in pitch; the diaphragm is depressed and the heart displaced unless adhesions prevent it. In left pneumothorax it may beat on the right side, the whole mediastinum being pushed to the right; in right pneumothorax the mediastinum may be pushed to the left nipple; hence there is resonance over the normal cardiac region. The pitch of the percussion note may be raised when the mouth is closed, and lowered when it is open (Wintrich's change of note), and a cracked-pot sound can be elicited in some cases, but this occurs only when the communication with the pleura remains open.

A valuable sign of pneumothorax is the coin test. A silver coin is laid upon the chest and struck by another, while the auscultator applies the stethoscope opposite to the point struck or over any part of the side distended by air. The ringing coin sound is reproduced with great intensity. It is pathognomonic, and the outlines of the cavity can be traced by it.

When fluid is present, as it usually is, there will be the ordinary signs of a pleural effusion, which have been sufficiently dwelt upon. The fluid is more mobile in pneumothorax, however, than in simple pleurisy, so that its level changes more quickly with change of posture of the patient, and *Hippocratic succussion* is readily obtained.

As the lung is compressed against the spine by the air, as it is by the fluid in pleurisy, the breath-sounds are feeble or absent, except over the root of the lung, where the breathing is bronchial. But if the lung is not completely collapsed, amphoric breathing may be heard, the air-chamber of the pleura acting as a consonance-box; it may be heard with both inspiration and expiration, or only with expiration.

Metallic tinkling is a sound believed to be due to the vibration of bubbling bronchial râles re-echoed through the air-chamber, or to drops of fluid falling from above upon the surface of the effusion. Re-echoing with metallic quality may also accompany the heart sounds, and in cases in which the respiratory murmur is amphoric the vocal resonance is of the same character. Vocal fremitus is generally absent. The prognosis of the affection depends upon the cause. Traumatic cases and those resulting from empyema are more favorable than those resulting from tubercle. In the latter cases death may occur suddenly from shock, or after a short time; or at times it may have a favorable influence upon the lung condition. Generally, however, it hastens death. When perforation has resulted from abscess or gangrene, the prognosis is very unfavorable, and in the latter case practically fatal.

Differential Diagnosis. *Pneumothorax* is most likely to be confounded with (1) emphysema; (2) tuberculosis of the lungs with large cavities; (3) cases of pleural effusion in which above the upper level of the fluid the lung is markedly hyper-resonant; and (4) abscess below the diaphragm containing air (pyo-pneumothorax subphrenicus).

Emphysema can be distinguished by its slow onset, its relatively slight impairment of the general health, by the fact that it is bilateral, whereas pneumothorax is almost always unilateral, and by the existence of feeble breathing with greatly prolonged expiration. Amphoric breathing and resonance, metallic tinkling, and signs of fluid are all absent in emphysema.

When the *pneumothorax* is circumscribed the physical signs resemble those of *pulmonary cavity*. But over a large cavity the chest is usually flattened; cracked-pot sound and alteration in pitch upon opening and closing the mouth are more common in cavity than in pneumothorax. Displacement of viscera does not occur in phthisical cavity, the coin test is negative, succussion cannot be produced. Fremitus is absent in pneumothorax and increased over a cavity.

The hyper-resonance above a *pleural effusion* develops with a very different clinical history, is accompanied by increase of fremitus with bronchial or, at times, amphoric breathing, and changes when the patient's mouth is open or closed. The percussion note usually loses the metallic quality heard in pneumothorax, metallic tinkling is absent, the coin test is negative.

Pneumothorax must be distinguished from abscess below the diaphragm containing air (*pyo-pneumothorax subphrenicus*). Often the distinction is difficult. Leyden points out the importance of remembering the sequence of events in the development of the disease. Where the abscess was situated below the diaphragm abdominal symptoms preceded its development, and early in the course of the disease there was absence of respiratory symptoms. Moreover, in subphrenic abscess the heart is not displaced or the interspaces bulging. In pneumothorax, according to Leyden, the respiration is normal, under the clavicle, and the transitions from the normal to the metallic and amphoric sounds lower down are abrupt. In pyo-pneumothorax on the left side the semilunar space disappears. In subphrenic abscess the amphoric sounds may be above and below the diaphragm or loudest at the epigastrium. In addition, in pyo-pneumothorax subphrenicus, as Mason points out, adhesions of the lung to the diaphragm and parietes may be made out, particularly if the case has been under observation in its earlier stages and dry pleurisy discovered. Abscess in this location and slight fluctuation are likely to develop with associated effusion. The very smallness in size of the effusion is of diagnostic import in favor of subdiaphragmatic inflammation.

CHAPTER III.

DISEASES OF THE HEART, THE BLOODVESSELS, AND THE MEDIASTINUM.

The Heart.

BEFORE the discussion of the symptoms and physical signs of heart disease is assumed, a brief review of some essential facts in the anatomy and physiology of the heart is of importance.

ANATOMY. The heart is a hollow muscle, composed of four chambers. The *muscle* is made up of unstriped fibre (involuntary muscle). The exterior is covered with a serous membrane—the *pericardium*, which is reflected upon the sac in which the heart hangs. The interior is lined with the same character of membrane, the *endocardium*. The *chambers* are four in number, two auricles and two ventricles. The auricles are at the upper portion or base of the heart; the ventricles at the apex. The heart is divided into two sides, the right and the left. An auricle and a ventricle of each side are related physiologically. The right heart draws blood from the veins and supplies it to the pulmonic circulation. The left heart belongs to the aortic side of the body, the major or arterial circulation, to which it sends blood, while it draws blood from the pulmonic circulation. *Valves.* The auricles are separated from the ventricles by valves named from their respective positions, the right and left auriculo-ventricular, or from their form the tricuspid and mitral valves of the right and left sides respectively. The valves close during the systole, producing the systolic sound, and open during diastole. The aortic and pulmonary valves are seated at their corresponding situations and close with the beginning of the diastole, producing the diastolic sound.

The heart receives its supply of blood from the coronary arteries and its innervation from nerve centres in the medulla, and from the sympathetic ganglia in the heart muscle.

TOPOGRAPHICAL ANATOMY. The form and position of the heart, its relation, and the relation of its anatomical elements to the surface of the chest, must be well known to understand the effects of disease upon its structure and function, and to recognize its physical alterations.

OUTLINE OF HEART ON CHEST WALL.[1] To have a general idea of the form and position of the heart, map its outline on the wall of the chest as follows:

(*a*) To define the base, *i. e.*, the part to which its great vessels are attached—draw a transverse line across the sternum, corresponding with the upper borders of the third costal cartilages; continue the line half an inch to the right of the sternum and one inch to the left.

[1] From Holden: Landmarks, Medical and Surgical.

(*b*) To find the apex, mark a point about two inches below the left nipple, and one inch to its sternal side. This point will be between the fifth and sixth ribs.

(*c*) To find the lower border (which lies on the central tendon of the diaphragm), draw a line, slightly curved downward, from the apex across the bottom of the sternum (not the ensiform cartilage) as far as its right edge.

(*d*) To define the right border (formed by the right auricle), continue the last line upward with an outward curve, so as to join the right end of the base.

(*e*) To define the left border (formed by the left ventricle), draw a line curving to the left, but not including the nipple, from the left end of the base to the apex.

Such an outline shows that the apex of the heart points downward and toward the left, the base a little upward and toward the right; that the greater part of it lies in the left half of the chest, and that the only part which lies to the right of the sternum is the right auricle. A needle introduced in the third, fourth, or fifth right intercostal space close to the sternum would penetrate the lung and the right auricle.

A needle passed through the second intercostal space, close to the right side of the sternum, would, after passing through the lung, enter the pericardium and the most prominent part of the bulge of the aorta.

A needle passed through the first intercostal space, close to the right side of the sternum, would pass through the lung and enter the superior vena cava above the pericardium.

The best definition of that part of the præcordial region which is less resonant on percussion was given by Dr. Latham years ago in his "Clinical Lectures." Make a circle of two inches in diameter round a point midway between the nipple and the end of the sternum. This circle will define sufficiently, or for all practical purposes, that part of the heart which lies immediately behind the wall of the chest and is not covered by lung or pleura.

Apex of the Heart. The apex of the heart pulsates between the fifth and sixth ribs, two inches below the nipple, and one inch to its sternal side. The place and extent, however, of the heart's impulse vary a little with the position of the body. Of this, anyone may convince himself by leaning forward, backward, on this side, and on that, at the same time feeling the heart. Inspiration and expiration also alter the position of the heart. In a deep inspiration it may descend half an inch, and can be felt beating at the pit of the stomach.

Valves of the Heart. The aortic valves lie behind the third intercostal space, close to the left side of the sternum.

The pulmonary valves lie in front of the aortic behind the junction of the third costal cartilage with the sternum, on the left side.

The tricuspid valves lie behind the middle of the sternum, about the level of the fourth costal cartilage.

The mitral valves (the deepest of all), lie behind the third intercostal space, about one inch to the left of the sternum.

Thus these valves are so situated that the mouth of an ordinary-sized stethoscope will cover a portion of them all, if placed over the sternal

end of the third intercostal space, on the left side. All are covered by a thin layer of lung; therefore we hear their action better when the breathing is for a moment suspended.

Action of the Heart. The heart beats, that is, alternately contracts and dilates or relaxes, 65 to 85 times per minute in an adult. The wide variation in frequency is accounted for by a difference in the number of beats in the two sexes. In females, the frequency varies from 75 to 85; in males from 65 to 75. With each beat, blood is propelled throughout the vascular channels of the body and drawn from them to the heart chamber. The first effect is produced by the contraction of the heart, or the *systole*; the second by the relaxation, or *diastole*. Other events, as the act of respiration, contribute to the completion of the outflow and the inflow of blood, particularly to the latter.

The completion of the act of contraction and the act of dilatation make up one revolution of cardiac action, or, as it is termed, a *cycle*. The act of contraction is known as the systolic period of the cycle; that of relaxation, the diastolic period.

Events of the Cardiac Cycle. Throughout the whole cycle several events occur. During the systolic period (1) the ventricles contract; (2) the auriculo-ventricular valves close; (3) the blood is squeezed from the ventricles into the vessels, and the columns of blood in the aorta and pulmonary artery receive a shock from the impact of the new volume of blood, and an increase in their bulk. The movement of the blood wave from this cause and the contraction of the large vascular trunks, produces pulsation of the peripheral vessels and the production of the *pulse*. The contraction is immediately followed by *relaxation*—the *diastole*. (1) The blood columns in the aorta and in the pulmonary artery fall back upon the valves guarding their outlets, the aortic and pulmonary valves. At the same time, (2) blood pours from the veins into the auricles. The filling of the the latter soon occurs. (3) The auricular muscles contract upon the blood in the chamber, driving it into the ventricles.

If a cardiac cycle is divided into tenths, the systolic period occupies four-tenths, the diastolic period six-tenths of the time. The systolic period occurs at the same time, or is synchronous with the apex beat and carotid pulse, and precedes by a fraction of a second the radial pulse. It is immediately followed by the diastolic period, which, therefore, follows the carotid and radial pulse.

Symptomatology. The symptoms of disease of the heart are due to the anatomical structure of the organ, to its physiological offices, and to the morbid process. The heart is a hollow muscular structure which hangs in a cavity and encloses cavities separated by valves. Both sets of cavities are lined by serous membrane. The serous membranes are subject to the same diseases and present the same symptoms that take place in serous membranes elsewhere. In inflammation of the external membrane the surfaces rub together and create a sound of friction. The external serous cavity may also become filled with the products of exudation or transudation. Physical signs are produced. They are, however, the physical signs of increased bulk of material as determined by inspection, palpation, and percussion, and made manifest by

physical interference with the heart. The heart muscle is also the seat of processes which obtain in muscular structures. They are hypertrophy and atrophy; inflammation, acute and chronic, with overgrowth of connective tissue; and degenerations. The symptoms correspond with symptoms of muscular hypertrophy or atrophy or of inflammation elsewhere, viz.: increase or weakening of the muscle, with, in the latter, the symptoms of the process. Increase or diminution in the power of the muscle is associated with corresponding change in size, which is determined by physical signs. Above all, however, such change modifies the functional office of the heart, so that strength or weakness of the muscle is shown in excess or deficiency of force of the circulation. The latter is more particularly an object of observation because of the congestions, dropsies, and cyanosis that ensue.

The heart is constantly subjected to internal pressure. Dilatation of the cavities or a portion of the cavity (aneurism) follows previous disease of the muscle or increase of internal pressure, and causes physical signs of enlargement. Degeneration of the heart muscle, nearly always secondary to deficiency of vascular supply, is also attended by symptoms of weakness and physical signs of enlargement (dilatation), or of diminution in size (atrophy). When dilatation occurs the orifices of the cavities enlarge. The valves cannot close them. Symptoms of incompetency and of blood regurgitation follow.

The serous membrane that lines the cavities of the heart, and with the subserous tissues makes up the structure of the valves, is subject to inflammations, the symptoms of which are common to all serous inflammations. The swellings and outgrowths that attend such inflammation occlude the orifices and prevent the closure of the valves. At each orifice obstruction to the flow of blood or improper closure of valves ensues. A physical interference with the heart's functions is produced, recognized by physical signs. The successful effort of the heart muscle to overcome such obstruction on the one hand (hypertrophy), or its failure on the other (dilatation), again leads to the production of symptoms and signs. The serous membranes, and hence the valves, are exposed to causes which excite inflammation. By virtue of the position of the heart at the head of the circulation, the blood, infected or irritating, as in rheumatism and Bright's disease, constantly bathes the vulnerable structure. For the same reason positive symptoms arise not common to serous membrane inflammation—that is, *embolic* phenomena (see Symptoms of Morbid Processes).

It is the function of the heart to propel the blood. It has been shown how interference with the action of the muscle and with the flow of blood through the cavities and orifices modifies the function. The functional power is increased or diminished by the physical changes. The evidence of increased power is seen in increased force of muscle and increased fluid wave in the arteries (pulse). Diminished power is shown in symptoms of lessened blood supply to parts, and in stagnation of the blood that is sent to the periphery. The former is more pronounced in cerebral anæmia, and physiological weakness of organs or the organism as a whole; the latter, in congestions and dropsies.

The functional activity of the heart is controlled by a nervous mechanism, any alteration of which alters cardiac action and consequently produces symptoms. Just as with the larynx, a break in the cardiac mechanism may be in the centres in the medulla, the centres in the muscle, or the sympathetic centres, or in the paths of the pneumogastric or sympathetic nerves to and from the heart. The rich anastomosis of these nerves exposes the heart to disturbance by reflex influences. We should suppose such extensive innervation would invite frequent cardiac perturbation. In a measure it does take place, but fortunately, in the perfection of this mechanism, the inhibitory fibres control such perturbation to a large extent, so that we do not see such pronounced symptoms as occur in the larynx. The symptoms which point to disturbance of the cardiac mechanism are alterations in the rhythm of the heart. Its action may on this account be increased or diminished in frequency, or it may be irregular or intermittent. Such alterations of rhythm may be due to organic disease of the centres, notably the pneumogastric from apoplexy, softening, or tumor in the medulla; to stimulation or depression of the centres by toxic substances in the blood, as in uræmia, acetonæmia or autogenetic toxins or other substances, or by nicotine or other extraneous material. The altered rhythm may be, and most frequently is, of reflex origin. It may be due to disease of the nerves, as the pneumogastric or sympathetic, from pressure upon the nerve trunk by tumor or inflammatory growth. Palpitation is the most pronounced symptom of altered rhythm of which the patient is cognizant. When it occurs, or other arrhythmical changes are found, the cause must be most frequently outside of the heart, and hence more frequently sought for beyond its domain.

While the symptoms or signs of cardiac disease are due to morbid processes in that organ or its membrane, it must be remembered that grave and persistent subjective and objective symptoms may be due to, or associated with, disease of contiguous structures outside of the pericardium. The symptoms are not excited through the nervous system, but are produced by mechanical encroachment upon the organ. They will be referred to in the study of objective symptoms. Care must be taken never to overlook the possibility of their presence.

In the study of the symptomatology of cardiac disease the student must bear in mind two things: first, that the causes of the morbid processes and of the symptoms (pain and palpitation) may be elsewhere than in the heart; and second, that the ultimate object of the examination is to determine the muscular power of the heart. He will soon learn that with that power intact the functions can be performed, notwithstanding the presence of marked physical abnormalities.

The recognition of disease of the heart is usually not attended by much difficulty, though some special lesions may involve difficulties in their determination. The non-recognition of cardiac disease is due to faults in the examination. The physician is too often satisfied with the recognition of the remote process, as a congestion or functional weakness in some organ. Safety lies, as has often been said, in the examination of all the organs of the body. Often, for instance, indigestion from gastric catarrh is not relieved because the cause, mitral disease, is not recognized.

The Data Obtained by Observation.

The objective signs of disease of the heart are determined by the same means as are employed in the detection of these signs elsewhere. In order to ascertain them it is necessary that the patient should be stripped, and a good light should fall directly on the surface as well as obliquely. The patient can be examined in any position, and indeed, for accuracy, should be examined in all positions. This is particularly true when the pulse rate is taken and when auscultation is practised. The sounds vary frequently in different positions, to the occurrence of which variation some diagnostic significance is attached. It is necessary sometimes to have the patient lean forward in order that the heart be brought more fully in apposition to the chest wall.

Inspection. Examination with the eye is not confined to the heart alone. The reader will remember that in the account of the examination of the exterior and local areas, abnormal conditions were pointed out, due to disease of the heart. In the examination, therefore, of a case of suspected heart disease, observation is made of the general color and color of the local parts, as the lips and the fingers, to determine the presence of *cyanosis*, *pallor*, *or jaundice*; of local areas, as the feet, to discover *dropsy*; the face, to note the appearance of the countenance; the neck, to note the state of the vessels; the eyes, to note their prominence; the thorax, to ascertain the presence of dyspnœa.

The Præcordia. The præcordia is the region of the chest which overlies the heart. In the study of the appearance of the præcordia, we observe (1) the degree of prominence of the chest in that region; (2) the appearance of the interspaces; (3) the hue of the surface; (4) the position of the apex beat; (5) the extent of the impulse. It may be unduly *prominent.* This is common in children who have had rickets and some possible cardiac hypertrophy in childhood. It persists in later life. The bony præcordia is prominent, irrespective of the soft tissues. The lower end of the sternum may project. This occurs in hypertrophy or dilated hypertrophy of the heart. In pericardial effusion, ribs and interspaces project. The latter are full or bulging even with the surface. In bulging præcordia the distance from the middle of the sternum to the mid-axilla is greater on the left than on the right side. Local bulging may be seen at the apex in cases of aneurism of the heart.

The præcordia may be *sunken.* Old pericarditis, but more frequently old empyema, causes sinking in of the region. It may be a result of *rickets* or of spinal curvature.

The *interspaces.* They are only retracted in pericardial adhesions; are full or bulging in effusion. Only when purulent pericardial effusion is about to rupture, or an empyema to discharge, do we note redness or other change in hue of the surface of the præcordia, not observed over the remainder of the thoracic surface.

The Apex Beat. The apex, or rather that portion of the heart which strikes the chest wall with each systole, is evident in health in the fifth interspace just inside of the mid-clavicular line. It can readily be detected by inspection in a good light in patients with moderately thick

chest walls. It is due to the impulse of the ventricle against the chest wall just as the muscle contracts, in systole. *Changes of position in health.* It is not a fixed point in health. It moves with the movements of the body, and hence when lying on the left side it falls toward the left axilla as far outward as the mid-clavicular line or even beyond that point. It moves toward the right and downward in full inspiration, or may disappear entirely toward the completion of that act. It may not be observed if there is a large amount of subcutaneous fat or if the mammary gland intervenes.

The Impulse. In health, in addition to observation of the position of the apex beat, the extent of the area of impulse is determined. This may be limited to the apex, or may be seen also in the third and fourth interspaces.

Change in the Apex Beat. The apex beat, or the lowest point of impulse, may be *displaced* or may be *absent* entirely. These changes are due either to (*a*) *disease outside of the pericardium*, to (*b*) *disease within the pericardium*, or to (*c*) *disease of the heart itself.*

Apex Displaced to the Left. This occurs from (*a*) *Alterations outside of the Pericardium.* The right lung may be the seat of extensive compensatory emphysema, on account of which the heart is dislocated to the left. Or the right pleura may be filled with large effusion, causing the same change in the heart. On the other hand, fibroid phthisis of the apex of the left lung, or pleural adhesions which have become attached to the pericardial sac, with, probably, coincident pericarditis, pull the heart to the left, thereby changing the position of the impulse. In disease of the mediastinum the heart is pushed downward and toward the left. An aneurism, an abscess, or enlarged glands in this situation may invade the normal cardiac territory and cause its dislocation.

In disease of the abdomen the apex is displaced. If the liver and spleen are enlarged, or the abdomen distended by ascites, the diaphragm is raised higher and the heart elevated. The apex is then seen to the left of the normal position, and may be one or two interspaces higher than natural. A common physical change in the stomach, dilatation, is a frequent source of dislocation of the apex. The dilatation may be temporary from flatulency or may be due to organic disease.

(*b*) *Alterations within the Pericardium.* In cases of pericardial effusion, the apex is said to be lifted to the left, upward and outward. It is seen in the fourth or even as high as the third interspace, and sometimes only an impulse is noted in the second interspace. If used in the sense that the apex is the most visible portion of the heart toward the left, the above is true; in other words, the edges of the left ventricle and the right ventricle, which make up the apex, are never tilted upward to the situations above mentioned. Instead, we undoubtedly see in pericardial effusions the pulse of the right auricle and the conus arteriosus against the chest wall.

(*c*) *Changes of the Heart Itself.* The apex is displaced to the left in dilatation and hypertrophy of the heart. In the latter it is also displaced downward. It may be as low as the sixth or seventh interspace and extend as far to the left as the anterior axillary or the mid-axillary line.

Apex Displaced to the Right. The same causes in general lead to displacement of the apex beat to the right. From (*a*) *Alterations outside of the pericardium.* We find the heart dislocated to the right in left pleural effusion, and in emphysema of the left lung. We find, moreover, in pleural contractions and in fibroid phthisis of the right lung, the heart drawn to that side. Under these circumstances the beat in its normal position is absent, and on inspection an impulse can be noted either in the epigastric region, along the margin of the ribs, or in the second or third interspace along the right edge of the sternum. The impulse in the epigastric region represents the hypertrophied right ventricle, which usually attends the lung changes that cause displacement of the apex beat. The impulse along the right edge of the sternum is certainly not due to the apex beat, but to the right auricle and the right ventricle brought in apposition to the chest wall by the cardiac dislocation. The apex, or the tip of the heart is, in all probability, displaced but little beyond the parasternal line. It may, theoretically at least, be pushed behind the sternum. (*b*) The apex beat is not displaced to the right in pericardial affections. (*c*) In disease of the heart itself there is no dislocation of the apex to the right.

Apex Beat Absent. Following the same order, we find that the apex beat may be absent entirely in (*a*) *disease conditions outside of the pericardium* which intervene between the heart and the chest wall. Hence, in emphysema of the lungs and in compensatory emphysema of the left lung, the apex, or indeed any cardiac impulse, is entirely effaced. (*b*) *In disease of the pericardium,* the apex beat is absent when there is a large effusion. The absence here succeeds the dislocation to the left, and with it occurs effacement of the impulse in the second and third interspaces. (*c*) *In certain diseases of the heart* the apex is absent entirely. This is due to diminution in the size of the heart, as in the atrophy of chronic disease, or of old age, or to diminution in the muscle strength of the heart when it is fatty or flabby from dilatation.

The Impulse. In health the impulse is limited usually to the area around the apex. The tissues of the thorax project with each systole. The area of impulse may be increased when the individual examined leans forward and at the end of expiration. It is larger when the chest walls are thin. It is lessened in opposite conditions. The area of impulse may be *increased.* The causes for increase in the extent of impulse are (*a*) *diseases outside of the pericardium.* This occurs in chronic phthisis with fibrous adhesions and in pleural adhesions. When the lung is drawn away from the surface of the heart an increase in the extent of the impulse is observed. When the heart is pushed against the chest wall it is also observed, as in aneurism or in diseases of the mediastinum, from inflammation or cancer, or other mediastinal growth. In the conditions above mentioned the impulse is seen not only in the third and fourth interspaces, but also as high as the second, and it is not limited to the spaces between the sternum and parasternal lines, but may extend beyond the mid-clavicular line. (*b*) From *disease of the pericardium* the area of impulse is increased if moderate effusion is present. It will be seen as a diffuse wave occupying the second, third, and fourth interspaces. It is also increased in pericardial adhesions without increase in

strength. (*c*) *Diseases of the heart.* The disease must cause enlargement, and hence must be either hypertrophy or dilatation. The extent of impulse varies. In hypertrophy the impulse may be communicated to the sternum, so that the lower part heaves with each contraction. It falls below the fifth interspace and toward the left, particularly if the left ventricle is the seat of the enlargement. If the right ventricle is hypertrophied the impulse is very marked in the sixth and seventh interspaces near the termination of the cartilages, or in the epigastrium along the border of the ribs of the left side. Sometimes, when associated with and displaced by lung disease, it is seen to the right of the xiphoid cartilage.

Impulse Absent. The same reasons that cause absence of the apex beat are sufficient to cause absence of impulse, and they need not be again repeated.

Retraction of Interspaces. In place of swelling or projection of the interspace or interspaces rhythmical retraction sometimes takes place. This retraction may be limited to the apex or may occur in each interspace over the præcordial region. It may occur with the systole or with the diastole. It is of some diagnostic significance when it is systolic in time, and is said to indicate adhesions of the pericardium, traction upon which by the systole of the heart causes the interspaces to be drawn in. The adhesions may prevent the lung overlapping the heart, so that the area of impulse and position of apex are not changed by full inspiration. (See Pericarditis.)

New Causes of Impulse. In addition to uniform increase or diminution in the impulse, new areas of impulse, not due to the extension of the normal impulse, arise from enlargement of one of the cardiac chambers or from disease of the bloodvessels. When seen in the second or third interspace on the left the area of impulse is due to hypertrophy and dilatation of the right ventricle, as in mitral obstruction; or it may be due to retraction of the lung in that region. If the impulse is noted in the course of the aorta or adjacent thereto it is indicative of aneurism. In the second or third left, or first or second right, interspace, the physical signs of this affection determined by palpation, auscultation and percussion indicate the nature of the pulsation.

Palpation. Palpation confirms inspection as to the shape of the præcordia, the condition of the intercostal spaces, the position of the apex beat, and the extent of the impulse. In addition we learn by palpation of the character and strength of the impulse, and note the presence or absence of valve shock and of thrills. By palpation also œdema of the surface is recognized and fluctuation may be detected. In a normal chest with moderate walls a slightly prolonged, moderately strong shock is transmitted to the hand when placed over the præcordia. It is synchronous with the cardiac and precedes the radial pulse. It is therefore systolic in time. It is stronger when the patient leans forward or exhales freely, removing the lung from the surface, or in thin chest walls it is weaker in opposite conditions.

Character and Strength of Impulse. A. The impulse may be *stronger* than in health. 1. Increased action. It must not be forgotten that in

the violent action of the heart that attends palpitation and the increased action in the early stages of fevers or of inflammation, the force of the cardiac impulse is much increased. 2. Disease. (*a*) On account of disease outside of the heart, by reason of which the heart may be brought nearer the hand. Increased force is also observed when the patient leans forward. If the lung is drawn away by adhesions or phthisical contraction, or the heart pushed against the surface of the chest by mediastinal growths, the force of the impulse is increased. (*b*) In pericardial adhesions the heart is held more firmly against the wall and may give the appearance of strength to the impulse. (*c*) True increase in force of the impulse is seen in disease of the heart itself. When the organ is hypertrophied or the seat of dilated hypertrophy, the force of the impulse is increased, sometimes for the patient to an almost unbearable degree. Uplifting of the præcordial area or even the lower half of the anterior part of the chest is seen. The hand or the head laid over the heart is forcibly lifted with each systolic contraction. This great force is most pronounced in the enormous hypertrophy that occurs in cases of aortic obstruction. It is the impulse and force of the so-called *cor bovinum*. In dilatation the impulse is diffused and wavy.

B The strength may be *lessened* or the impulse not felt at all. This occurs from causes which diminish the impulse or cause it and the apex beat to be absent entirely, as when material intervenes between the heart and the chest wall, or the heart is weakened by disease. Hence (*a*) in emphysema of the lung; (*b*) in pericardial effusions; (*c*) in fatty heart, or myocarditis, in dilatation and simple weakness of the heart, the strength of impulse is lessened.

Valve Shock. The shock of the closure of the valves can be felt by the hand when placed evenly over the præcordia. The shock from the pulmonary and aortic valves is best transmitted. It is felt most distinctly in persons with thin chest walls and in cases in which there is heightened tension either in the aorta or pulmonary artery. The shock follows the impulse. It may be localized more precisely by the finger-tips in the third or fourth interspace along the left edge of the sternum. The shock of the auricular ventricular flaps is also transmitted. The shock is synchronous with the first sound. It is felt in the left fourth interspace near to the sternum, sometimes over it. It is due to dilatation of the heart and is more readily felt in thin-chested persons.

Thrills. A thrill is produced when the blood is thrown into vibrations by passing over a rough surface. It may be created with the systole or during the diastole. It can only be created at the time blood is passing through the orifices. 1. The most common seat of the thrill is at the apex. If the hand is placed in close apposition to the surface of the chest at this point a vibration or tremor is transmitted to it in most cases of *mitral obstruction*. The blood is passing from the auricle to the ventricle; as this takes place before the systole the thrill is felt at that time and hence before the impulse or carotid pulse. It is *presystolic* in time. It is sometimes difficult, however, to separate it from the impulse. Its characters cannot well be described. The hesitating, jogging manner of the vibrations or the thrill is clearly transmitted to the hand. 2. The next most frequent seat of thrill is at the second

costal cartilage on the right. Here the thrill or vibration is *systolic* in time and is caused by *obstruction* at the *aortic orifice.* It may be felt away from the heart, in the aorta, or in the carotids. The aortic valves are thickened, contracted, and stiffened by a sclerotic endocarditis or the orifice occluded by valvulitis. 3. Sometimes a thrill is felt at the apex with the systole—*first sound.* This occurs rarely, but must not be confounded with the before-first-sound thrill. It is never so distinct, and is not made up of a series of vibrations. It is due to regurgitation at the mitral orifice. 4. Rarely a thrill is felt at the second costal cartilage on the right, with the *second sound.* It may be felt along the course of the sternum also, and is due to regurgitation through the aortic orifice. The systolic thrill must not be confounded with the thrill elicited over the aorta or at the aortic cartilage due to aneurism. 5. At the second costal cartilage at the left a thrill is sometimes felt. It is *systolic* in time and is not transmitted. It is due to obstruction at the pulmonary orifice. 6. At the lower portion of the sternum a thrill systolic in time is also felt, due to tricuspid regurgitation. Care must be taken not to confuse the above-mentioned thrills with those due to aneurism. (See Aneurism.)

Pericardial Friction. In addition to the thrills a friction or to-and-fro rubbing is transmitted to the hand in cases of pericarditis in the first stage. The friction is felt over the heart region, but is pronounced in the third or fourth interspace. It may be detected on slight pressure or not revealed unless firm pressure with the tips of the fingers in the interspaces is used.

It is important to remember that the *position* of the patient modifies or weakens the intensity of thrill or friction. When the patient is lying down it may not be felt. The upright posture or leaning forward makes it evident, and hence the patient should be instructed to assume this position in the examination if possible.

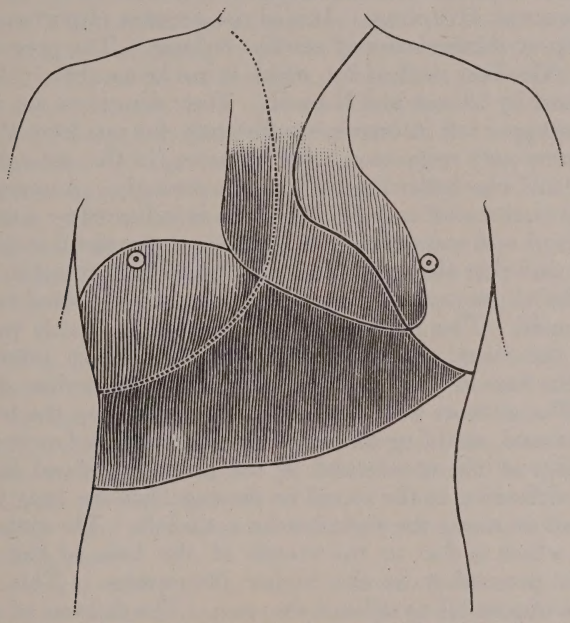
Diagnostic Significance. Of the above-mentioned thrills the one due to mitral obstruction is more frequently present in that valve lesion than thrills in other lesions. Indeed, it is pathognomonic of the disease.

Percussion. By means of percussion the form and size of the heart and the area of cardiac dulness are determined. The lungs overlap the heart and in inspiration allow a small area to be in apposition with the chest wall. To determine the size of the heart, both superficial, or light, and deep, or strong, percussion must be employed. By the former we determine the area of superficial or absolute cardiac dulness; by the latter, the area of deep cardiac dulness.

1. The Area of Superficial or Absolute Cardiac Dulness. —It is the area not covered by the lung at the time of inspiration. The percussion force employed must be light, so as to elicit the resonance of the extreme thin edge of the lung. The area extends from the fourth to the sixth costal cartilages. The right border may be defined by a line drawn between two points fixed on the median line of the sternum opposite the cartilages above indicated. Join the upper extremity with a point at the position of the apex beat. It marks the upper and left border.

A line joining the apex and the point on the median line of the sternum opposite the sixth costal cartilage and above the ensiform cartilage, marks the lower border.

FIG. 59.



Showing percussion of the heart and liver, the degree of shading indicating the degree of dulness. The margin of the lung is indicated by the dotted lines. The liver is enlarged. (GIBSON and RUSSELL.)

Method. The right border is determined by percussing from without inward to the median line. Always begin to percuss far enough from the sternal edge to get the clear pulmonary note. To insure uniformity, select a definite area in all cases from which to start. Apply the finger vertically at first. The right border may correspond with a line outside of or along the right edge of the sternum, the median line or the left edge of the sternum, or even beyond the latter. After the edge of the modified resonance is reached, percuss with the finger parallel with the ribs, as described by Gibson, to control the result previously secured, and as each interspace is percussed to determine the upper limit of liver dulness and the triangle between the liver and heart.

The left edge is determined by percussing in vertical lines from a point near the axilla toward the heart. Opposite the second and third interspaces on the right side the aorta, and on the left the pulmonary artery, can be defined. The student should acquire the habit of proceeding from definite fixed positions toward the heart, and to observe the changes during inspiration and expiration. The lower border and rounded apex cannot be defined if the stomach contains food or fluid. It is triangular in shape, with the apex pointing downward.

Changes in Size. The superficial area of dulness or absolute dulness is *increased* in pericardial effusion or enlargement of the heart. It is

replaced by resonance in emphysema, and hence *absent* entirely, as the lung overlaps or completely covers the heart. It is absent when the heart is drawn under the lungs by adhesions, and when there is air in the pleural or pericardial sac.

Deep Cardiac Dulness. It is of the greatest importance to ascertain the deep or *relative area* of cardiac dulness. The percussion must be strong. The best method by, which it can be accurately determined, is that advised by Gibson and Russell. Their directions are as follows: Begin in the upper left interspaces sufficiently far out from the sternum to secure pulmonary resonance. For instance, in the second interspace begin in the mid-clavicular line and percuss strongly. As soon as a slight alteration in that sound is noted the point is indicated by a mark. The second or third and succeeding interspaces are percussed in like manner, bearing in mind that the percussion must begin farther out in each interspace in order to get pure resonance. As dulness is secured in each space a mark is made. This is continued to the apex if that is visible, or to the base of the chest. By joining the marks in each interspace with the line at the base of the heart the left border of cardiac dulness can be fixed. The authors well point out that in this way the true apex of the heart is found, enabling auscultation to be conducted more accurately. The right edge of the vessels and of the heart is defined in the same way. The difference in the sound in passing from the lung to the heart is not so distinct along the right border as the left. The authors include the dulness which is due to the vessels at the base of the heart, and hence begin percussion in the higher interspaces. This is proper, because it is impossible to delimit the two. The dulness of the vessels is not so marked, however, and may be indicated by simple change in pitch in the percussion note. The lower border of cardiac dulness is ascertained with difficulty, because of its close apposition with the liver. At times there is a difference in the character of the dulness between the two organs. It can be well made out by stethoscopic percussion. This may not be so pronounced as we pass from the heart to the liver in the median and parasternal lines. Toward the apex the difference is more apparent. The writer has been teaching and practising this method of percussion ever since it was proposed by the authors, and can testify to its accuracy in clinical studies and the ease with which students are able to practise it.

Deep Dulness Increased. The increase in the area of relative dulness in all directions occurs in hypertrophy of the heart and in pericardial effusions. The increase in width above the base of the heart occurs in dilatation and aneurism of the aorta. Change in the position of the heart, a general idea of which is previously obtained by inspection and palpation, always changes the shape and extent of the dulness. The heart should be accurately delimited when displacements have taken place.

Increase or Extension of Deep Dulness Upward or to the Right or Left. In addition to general increase in cardiac dulness, one of the boundaries or a portion of the boundary may be increased or extended beyond the normal line. (1) Thus the area of dulness may extend *upward*. It may be followed by extension of the right and left boundaries. The relative

area of dulness is abolished. The change from pulmonary resonance to dulness is decided. The heart dulness becomes pyramidal or pyriform in shape. It is due to effusion in the pericardium. (2) Increase in dulness to the *left* occurs in enlargement of the heart from hypertrophy or dilatation. If the dulness extends outward to the left and retains the triangular shape, with the apex pointed, it is due to hypertrophy of the left ventricle. If, on the other hand, it becomes quadrilateral in shape, with the apex rounded, it is due to dilatation of the left ventricle. The results of palpation and inspection aid in proving the presence of one or the other of the two conditions. (3) The area of dulness extends to the *right*. It is due to hypertrophy and dilatation of the right auricle and ventricle. When the auricle, the right edge is extended beyond the normal in the third and fourth, or as high as the second, interspace. With this increase in dulness there is also seen an epigastric impulse, venous turgescence and pulsation of the veins of the neck or of the liver. (4) Increase in the area of dulness over the bloodvessels is usually due to aneurism. It may be general, as in dilatation, or more marked in local situations. Extension of the dulness outward or upward from the normal line may be found at the right of the sternum, or over the first bone of the sternum, or to the left just above the cardiac area. In the latter the dulness is an extension upward of the normal area of cardiac dulness with rounding of the area affected. The aneurism is situated in this case at the commencement of the aorta.

Pleximetric Percussion. For more accurate cardiac percussion, Sansom recommends the use of a pleximeter of his design by which delicate shades in dulness can be readily detected by the ear. The pleximeter is a thin, flat, oblong plate one inch by half an inch, which has on its upper surface a column rising from the middle, one and a half inches in height, which is surmounted by a plate three-fourths to three-eighths of an inch set parallel with the lower plate. The instrument is held by the fore- and middle fingers of the hand, applied on each side of the vertical column, the sensitive tips of the fingers resting on the upper surface of the larger horizontal plate. The lower surface of this latter is applied closely to the wall of the chest, and percussion with one or two fingers of the right hand with an even and not too forcible stroke from the wrist is made upon the upper plate. Of course vibrations are created by the blow. They are transmitted to the ear and also appreciated by the digital sense of touch, both of which aid in the determination of the nature of the sound produced.

Method. The pleximeter is placed with its long diameter parallel with the sternum about midway from the axilla to the right sternal border. Percussion is made upon the summit of the column by one or two fingers. As this is performed the pleximeter is moved, always in parallel lines, nearer and nearer to the sternum. A line is reached where the vibrations are modified. Incline the pleximeter so that the vibrations come from its left edge. This edge, or line, is practically the line of demarcation of the dulness and should be indicated by an aniline pencil. It corresponds to the outline of the right border of the heart. (See Fig. 59.) The process must be repeated at higher and lower levels

until the entire right area of cardiac or aortic dulness is ascertained. In passing, it may be stated that percussing from above downward with the long diameter of the pleximeter horizontal instead of vertical, leads to the upper limit of the liver as indicated by modified vibrations. About the fifth right intercostal space a short curved line is thus made out along the right edge of the sternum which indicates the outline of the right auricle at the point where it joins the liver dulness. Above this, as far as the second rib, the line indicates the outline of the right border of the auricle and the aorta. The outline of the auricle may be in the mid-sternum ; of the aorta, at the right edge. In percussing the left side of the chest the same method is adopted. Begin at the level of the second rib two or three inches beyond the left edge of the sternum, and move to the right. Join the lines of modified vibrations, and in this manner the left border of cardiac and aortic dulness is secured. The outline of the apex of the heart is readily mapped out. Over the tympanitic stomach light percussion is necessary. To narrow the area of percussion about the apex the percussion may be performed on the larger plate while the smaller is applied to the chest. The vibrations over the liver and over the right ventricle are difficult to distinguish, although sometimes so different that demarcation of the border of the ventricle presents no difficulty. Between the apex of the left ventricle and the left lobe of the liver the space is easily marked out. A correct outline of the heart and of the vessels is thus obtained. The upper limit of dulness is formed by the right auricle, the aorta and the pulmonary artery. Any bulging or undue expansion is due to aneurism, or aneurismal dilatation of the aorta. The space between the apex and the left lobe of the liver defines the lower border. Sansom well points out that by his method of percussion the following absolute data can be obtained : A projection to the right of the area of the upper part over the second and third interspaces, points to aneurism of the aorta or of the innominate artery. It may be traced to the left side of the sternum on account of saccular dilatation of the aorta. If the dulness at the upper part extends greatly to the left an increase in size of the pulmonary artery may be suspected. Along the mid-sternal region, extension beyond the right side joining the line indicating the upper border of the liver indicates distended inferior cava. This distention occurs in right-sided dilatation of the heart, and the dulness may also be due to dilatation of the adjoining auricle. The outline of dulness obtained over the apex of the heart if pointed indicates hypertrophy ; a more rounded outline shows dilatation. In uncomplicated hypertrophy the line of the right ventricle forms a much less obtuse angle with the liver dulness than in dilatation. Of great diagnostic value is the diminution of the area of dulness from atrophy of the heart as observed in wasting, as in cancer and in tuberculosis ; it may also be observed in typhoid fever. In the above-mentioned conditions it is a bad prognostic sign.

Adjacent Dulness. Care must be taken not to confound the dulness of pleural effusion or consolidated lung with the cardiac dulness.

Repercussion. Modification of the vibrations felt by the fingers on the pleximeter, as pointed out by Sansom, may indicate an abnormal change in physical condition not attained in any other way. It

is to be remembered that over the lungs the vibrations are in excess; over solid structure they are modified or lessened. Now, change from vibrations to absence of vibrations may be gradual or abrupt. Sansom determines as follows by repercussion after the heart has been outlined in the above-mentioned manner. In percussing from the lung and the heart area, if the modified vibrations occur abruptly, it is very probable that there is pericarditis with effusion or thickened pericardium. Or if, on percussing from above downward, there is effusion in the pericardial sac, no vibrations are to be elicited over the area delimited. That is, the absence of vibrations is noted over the whole area; whereas, in ordinary conditions, when the pericardium is unaffected, in percussing from above downward over the area which had been delimited on the right and left sides respectively, a line will be reached where the vibrations become modified. This line commences a little above the ensiform cartilage and inclines toward the left border of the cardiac dulness at the level of the fourth rib and the third interspace. Vibrations are more marked above than below the line. The line at which the lessened vibrations begin points out the commencement of the thick wall of the ventricles; the portion above (more vibratory) indicates the position of the right auricle and vessels. If plessimetric percussion is employed, areas of superficial and deep dulness need not be estimated.

Apex Beat. Whichever method of percussion is employed it will be often observed that the spot noted by inspection and palpation as the apex beat is far outside of the left border of cardiac dulness. In hypertrophy of the left ventricle it may be a considerable distance to the left. In dilatation the difference is not so marked. The percussion lines are made when the heart is away from the chest, and hence are within the systolic apex beat.

Method of Graphic Record. We are indebted to Sansom and Ewart for a method of record of the outlines of the areas of dulness and the position of the apex beat and other pulsations, which is of great value for class demonstration and for permanent records in order to compare with other records taken from time to time. The points of pulsation and border lines of dulness are marked by a dermatographic pencil. Various colors may be used in order to indicate the different data. The landmarks, etc., are outlined by a camel's-hair pencil dipped in olive oil. The episternal notch, the clavicles, the intercostal spaces, the ensiform cartilage and nipples, etc., the percussion outlines, and other recorded marks, are passed over with the pencil. A sheet of tissue paper, or of copying paper, is then gently pressed over the whole, so that the oil-marks are imprinted. After the paper is removed, the oil outline is colored by the dermatographic pencil, and a permanent record is preserved. By this plan of recording a maximum of precision is attained. Outlines can be measured and positions defined by mathematical data. The name of the patient, the date of observation, with a brief history of the case, should be attached to the chart. If the colored pencil-markings on the patient's chest are objectionable, the outline may be made with the colorless oil-pencil at the various steps of the examination. After they are transmitted to the paper they may be made more graphic by the colored pencils.

As an objection to the use of the above for comparative records, Ewart has shown that after long intervals the size of the chest and abdomen are apt to alter from various circumstances—growth, muscular development, habit of sitting, etc. He points out the advisability of using a fixed structure for reference, as the sternum, which is invariable. By utilizing its edges we have unalterable landmarks.

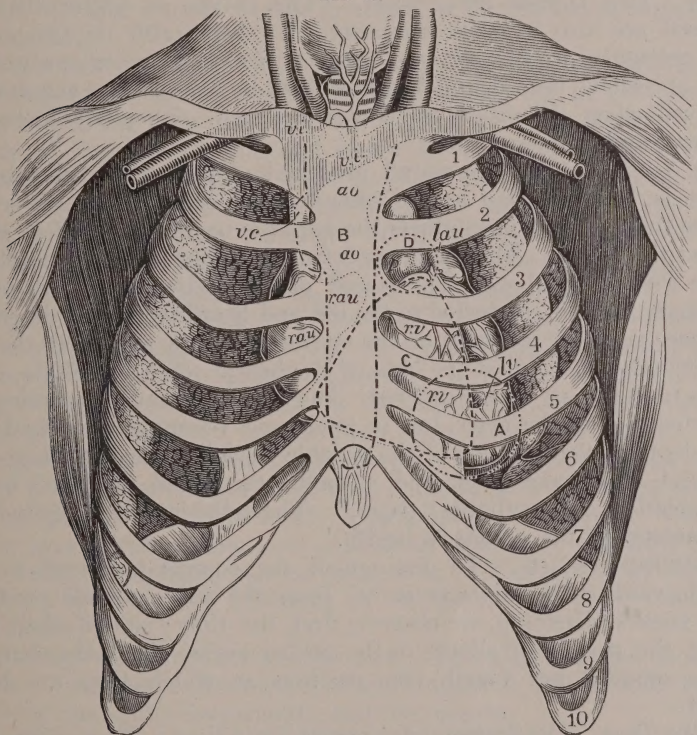
Sense of Resistance. Ebstein delimits the heart by the sense of resistance, change in size being noted by increase or diminution of the area, which in health gives a sense of resistance to the percussing finger.

Auscultation. *Method.* Either method of auscultation may be employed in order to secure data derived from the sense of hearing. The mediate, however, is preferable because it is essential to localize the sounds that are heard. By the immediate method we may form a general notion as to the condition of the heart sounds, but for the above-mentioned reason, and because if the double stethoscope is used we can also inspect the cardiac area, auscultation by the mediate method is preferable. The patient should be in a comfortable position. The muscles should not be strained. The general direction for performing auscultation must be followed out. Before it is commenced the observer has determined, if possible, the presence of the apex beat. If not, the first step must be to find the radial or carotid pulse. By this means the events of the cardiac cycle are ascertained. The systole is synchronous with the apex beat, or carotid pulse. It occurs just before the radial pulse.

The Sounds in Health. The stethoscope is placed over the base of the heart at about the fourth interspace, with the finger on the apex or the radial pulse; a sound will be noted corresponding with the systole or apex beat, followed almost immediately by another sound and then a period of silence. The sound that attends the systole is known as the systolic, or *first sound.* The sound that follows is known as the diastolic, or *second sound.* The sounds and silence mark the completion of a cardiac cycle as far as the ear is concerned. A definite relationship in time exists in the cardiac cycle. If the entire cycle occupies one second of time and is divided into tenths, the sound that attends the systole will occupy four-tenths, the interval between this first sound and the one in diastole, one-tenth; the sound that attends the diastole, two-tenths; and the silence, three-tenths of the entire period. By the above method, the first essential in auscultation is learned, viz., to associate apex with pulse beat and the relation of the sounds to the sounds of a cardiac cycle. In this manner the rhythm of the heart is ascertained and the character of the sounds is then studied. The character depends upon the cause, the points of origin and direction of conduction. *Cause.* Four sounds are created during a cycle, one at each valve. The sounds created with the systole (systolic sound) are due to contraction of the right ventricle and closure of the tricuspid valve; and on the opposite side, of the left ventricle and the mitral valve. The rush of blood along the course of the vessels and the shock of the heart may contribute somewhat to the systolic sound. The sounds heard in the beginning of the diastole are due to the closure of

the aortic and pulmonary valves. They are due to the tension produced on the valves as the respective arteries contract upon the columns of blood. The closures of the valves make up most, if not all, of the sounds. To review: two sounds occur with the systole, one from closure of the mitral, another from closure of the tricuspid valve; two with the diastole from closure of the aortic and pulmonary valves respectively. Modifications in the intensity of the sound are due to changes in the tension of the valve curtains, and are dependent upon the muscle. If it is strong, the valves are made more tense. Experiment and the results of disease have aided in proving these points.

FIG. 60.



Areas of cardiac murmurs (Gairdner for the areas; and Luschka for the anatomy). The outlines of organs, which are partially invisible in the dissection, are indicated by very fine dotted lines; while the areas of propagation of valvular murmurs, as described in the text, have been roughly marked by additional much coarser and more visible dotted lines—the character of the dots being different in each of the four areas. A capital letter marks each area, viz.: A, the circle of mitral murmurs corresponding with the left apex; B, the irregular space indicating the ordinary limits of diffusion of aortic murmurs, corresponding mainly with the whole sternum, and extending into the neck along the course of the arteries; C, the broad and somewhat diffused area occupied by tricuspid murmurs, and corresponding generally with the right ventricle; D, the circumscribed circular area over which pulmonic murmurs are commonly heard loudest.

Reference letters: r. au. = right auricle; a. o. = arch of aorta; v. i. = the two innominate veins; v. c. = vena cava descendens; p. = pulmonary artery; l. au. = left auricle; l. v. = left ventricle; r. v. = right ventricle. (FINLAYSON.)

Seat of origin and transmission. The sounds produced by the closure of the valves are developed, as the topography of the heart

shows, quite near to each other, but by the conduction of sound they are transmitted in particular directions, and heard loudest in definite areas on the chest. *The systolic or first sounds.* The *mitral area.* The sound produced by the closure of the mitral valve created at the fourth interspace near the sternum is transmitted to the surface of the chest by the thickened left ventricle, and hence is heard loudest where that is nearest the chest, namely, the apex. The *tricuspid area.* The sound produced by the closure of the tricuspid valve is transmitted by the right ventricle and is heard loudest over the lower portion of the sternum. Thus it is seen that the systolic, or first sounds, are heard loudest at the lower portion of the heart. *The diastolic or second sounds.* Two sounds are created. The valves at which they are produced are also in close proximity. To distinguish the two sounds it is necessary to auscult over areas to which they are transmitted. These areas have been definitely ascertained by the same means as those employed when the other valve was analyzed. They are known as the aortic and pulmonary area.

The Aortic Area. The sound produced at the aortic valve by its closure is heard loudest at the second costal cartilage on the right, because the aorta which conducts the sound is nearest the surface of the chest at this point. This cartilage is known as the aortic cartilage.

The Pulmonary Area. The sound produced by the closure of the pulmonary valve is conducted to the left and heard loudest in the second interspace near the left edge of the sternum. It is seen that the diastolic sounds are heard at the base of the heart. (See Fig. 60.)

Character of the Sounds. The systolic sounds are prolonged, somewhat dull in character, low in pitch, and resemble the sound produced by the pronunciation of the syllable "*ubb.*" The diastolic sounds are short, sharp, and quick and resemble the sound produced by the pronunciation of the syllable "*dupp.*" The syllables *ubb*, *dupp* indicate the character of the sounds in health.

Differentiation. To distinguish the sounds produced by the auriculo-ventricular valves (systolic) from the valve sounds produced at the vessels (diastolic), we observe, first, the time; second, their relation to the periods of silence in the cardiac cycle; third, the character of the sound; and fourth, the position at which they are heard loudest.

1. *The Time.* The first sounds are systolic in time. They occur at the same time as, and correspond with, the apex beat and carotid pulse, and they precede slightly the radial pulse. They are followed by a short silence. The second sounds are diastolic and follow the pulse.

2. *Relation to the Period of Silence.* The second sounds practically follow the first and precede the long silence.

3. *The Character.* The first sounds are low in pitch, dull and prolonged; the second sounds are high in pitch, short and sharp.

4. *Situation.* The first sounds are heard loudest at the apex of the heart and the base of the sternum and are transmitted toward the axillæ. They may be heard all over the cardiac area, but the position of maximum intensity is in the lower portion and toward the left. The second sounds are loudest at the base of the heart. They may be propagated

beyond the præcordia toward the neck and be heard loudest in the vessels of the neck.

Differentiation of Each Sound. 1. *Mitral* first or systolic sound, heard loudest at the apex, inward to the parasternal line, upward to the third interspace. 2. *Tricuspid* first or systolic sound, heard loudest at the lower part of the sternum and toward the left to the parasternal line as high as the third rib. 3. *Aortic* second or diastolic sound, heard loudest at the aortic cartilage, propagated into the vessels and also heard at and outside of the apex beat. 4. *Pulmonary* second or diastolic sound, localized to the second interspace and the third rib.

Modifications of the Sounds. All of the sounds or one or more of the four sounds may be increased or diminished in intensity or accentuation.

All Sounds Increased. *a.* Causes outside of the pericardium. (1) Anything which brings the heart closer to the ear of the observer. Thus, in patients with thin chest walls, when the heart is pushed to the surface of the chest (mediastinal tumor) or the lung removed (pleural contraction). (2) Anything which conducts the sounds, as consolidated lung in the vicinity, or a pneumothorax, or pulmonary cavities; the sound is intensified. *b.* Affections of the pericardium, as pericardial adhesions. *c.* Conditions of the heart. (1) Hypertrophy. (2) Overaction, as in palpitation, anæmia, fevers, exophthalmic goitre.

Weakness of All Sounds. *a.* Conditions outside of the pericardium. 1. General exhaustion. 2. Thick chest walls, large mammary gland. 3. Emphysema of the lungs overlapping the heart. *b.* Conditions in the pericardium, as fluid or air in the pericardial sac. *c.* Conditions of the heart. Atrophy; myocarditis; some cases of dilatation.

In short, *loudness* of all the sounds occurs from (*a*) conditions outside of the heart: heart nearer chest wall, consolidation of lungs, cavities; (*b*) conditions of the heart itself: hypertrophy; overaction. *Weakness* of the sound occurs from—(*a*) conditions outside of the heart: thick chest walls, emphysema, general exhaustion; (*b*) affections of the pericardium: effusions; (*c*) affections of the heart: atrophy; dilatation; myocarditis.

Changes of Individual Sounds. The above applies to all the sounds. Increase or diminution of the systolic or the diastolic sounds, or of any one of the four sounds, may be present.

Increase in Loudness of the First Sound. Increased loudness of the first sound is noted when the muscle is hypertrophied, and the tension on the valves thereby increased. In hypertrophy of the left ventricle the increase is most marked. The sound is duller and has a prolongation which is very characteristic. In hypertrophy of the right ventricle the sound is dull and prolonged over the sternum, but not to the degree of the left when it is hypertrophied.

Increase in Loudness of the Diastolic Sound. Either of the second, or diastolic sounds, may be increased in loudness or accentuated.

Accentuation of the Aortic Diastolic Sound. Anything which causes increased tension in the aortic circulation, and hence increased contraction of the aorta, will increase the intensity or accentuation of the second sound. In hypertrophy of the heart the aortic sound is accentuated

because there is corresponding increased contraction of the aorta following the forcible propulsion of the blood from the ventricle. Increase in arterial tension is due to increased contraction of the aorta when there is peripheral resistance to the outflow of blood. It is associated with the following conditions which cause accentuation of the second sound: Atheroma of the aorta, or of the arteries in general, is attended by increased accentuation of the second sound when there is at the same time heightened arterial tension. It is present in aneurism of the aorta. It is notable in disease of the kidneys, and particularly in that form in which there are also general arterial changes, namely, chronic interstitial nephritis. It is true that a portion of the accentuation may be due to the hypertrophy of the heart which exists.

Accentuation of the aortic second sound occurs independently of permanent change in the arteries. If for any reason there is spasm of the peripheral capillaries, as from a chill, from epilepsy, from nervousness due to hysteria, tension in the arteries is heightened, and hence the second sound accentuated. It is seen that accentuation of the second sound is therefore a marked index of the state of the vascular system in general; it is not only an evidence of disease of the heart. In certain fevers and in states of the blood in which the vasomotor nerves are irritated, causing peripheral contraction, as in scarlatina, accentuation of the second sound follows. This arises often before the development of local inflammatory diseases due to the same cause, as nephritis in scarlatina. The occurrence of this complication may be suspected when accentuation of the aortic second sound is heard.

Accentuation of the Pulmonary Diastolic, or Second Sound. This is due to the same physical condition which causes accentuation of the aortic second sound. Anything which heightens the tension in the pulmonary artery will cause increased loudness. In health the pulmonary second sound is not so loud as the corresponding sound of the aorta. If, therefore, we find in the second or the third left interspace the sound as loud as an aortic sound, or louder, it can be said that the pulmonary second sound is accentuated. It is due: (1) To any condition which causes congestion within the lungs, the right ventricle being at the same time of normal or increased strength. It is heard in the early stages of pneumonia, and if the course of the disease continues favorable may remain accentuated to the end. If, on the other hand, the circulation is embarrassed, and the right heart is failing, it will become fainter, and may be scarcely recognizable. Such change in the sound accompanies increase of respiratory distress, and indicates that the right heart is becoming exhausted. It is a sign of prognostic omen in acute pulmonary disease. If the case is unfavorable, the signs of right-sided dilatation will subsequently occur. (2) It occurs in emphysema of the lungs. Notwithstanding the covering of the heart by the lung, the sound can be heard, and may be the only one of the four sounds which is distinguished. (3) In valvular disease of the heart seated at the mitral orifice, accentuation of the pulmonary second sound, due to increased tension in the artery, is heard. In mitral obstruction the blood is retained in the auricle and pulmonary veins, causing a pressure, which is exerted against the force of the right

ventricle. Increased tension in the pulmonary artery is a result, with exaggerated strain upon the valves. In mitral regurgitation, with the systole the blood is thrown back into the auricle, and consequently meets with blood coming from the lungs. This in time increases the amount of blood and of blood pressure in the pulmonary artery. A heightened tension results.

Skoda pointed out the significance of this association. Sometimes in doubtful cases, either in the presence or absence of a murmur at the mitral orifice, the occurrence of this sign makes it more than probable.

Diminished Accentuation or Weakness of the Aortic Sound. This is an indication of cardiac weakness, and is liable to ensue in the course of fevers when exhaustion takes place. It is a sign of myocarditis and of degeneration of the muscular walls of the heart. Under these circumstances the systole of the ventricles is also weakened.

Feebleness of the aortic second sound, with a strong systole of the ventricle, occurs when the aortic leaflets are swollen or enlarged and thickened. This condition of the valves is due to atheroma, and is in all probability associated with atheroma of adjacent vessels, as the coronary arteries. It is, therefore, a sign of serious import.

Diminished Accentuation or Feebleness of the Pulmonary Sound. This is of importance to note in the course of valvular disease of the heart, providing previous accentuation has been observed. If the marked loudness gives way to feebleness there is strong probability that the right heart is undergoing dilatation with regurgitation at the tricuspid orifice. While accentuation of the pulmonary second in valvular disease is of good omen, enfeeblement of the sound is of bad prognostic omen, indicating weakness of the right ventricle.

Feebleness of the Mitral Sound. Feebleness of the mitral sound observed at the apex of the heart may be an indication of weakness of the muscle from dilatation, atrophy or myocarditis. It must be remembered, however, that weakness of the ventricle is not attended by enfeeblement of sound, but that when the right or left ventricle is dilated the duration of the sound is lessened. The loudness remains the same, or may be increased. Note, then, that a short systolic sound, loud, sharp, flapping, heard at the apex, indicates dilatation or feebleness. The tension of the ventricles and valves creating the sound is increased by internal pressure. The systolic sounds become like the diastolic, and may be distinguished from them with difficulty. With the finger on the apex or carotid artery, if the heart's action is slow the first sound will correspond with either pulsation.

Alterations in the Rhythm. *Fœtal rhythm* of the heart: Embryocardia—a term first used by Huchard to designate a condition in which the pause between the heart sounds is of equal length. The first and second sounds are exactly alike, resembling the beat of the fœtal heart. The sign is of importance in prognosis. In acute disease and in fever it indicates enfeeblement of the heart and reduction of arterial tension. In the later stages of Graves' disease it is a forerunner of death. It is distinguished from the rapid beat of the heart in tachycardia by the fact that in the latter condition the normal rhythm is preserved.

Cantering Rhythm of the Heart. The ear recognizes three sounds. The usual sounds may or may not be attended by murmur, and the interpolated sound may be dull or short and sudden. It may occur at various periods in the cardiac cycle, either before the systolic sound, after the diastolic sound, or during the diastolic pause. The rhythm recalls the sound of a horse cantering. It was termed by Bouillaud the *bruit de galop.* When the interpolated sound resembles the first or the second it is similar to reduplication of the sounds. It has been observed in hypertrophy of the heart, especially of the left ventricle; dilatation of the heart; in adherent pericardium, with dilated hypertrophy; in myocarditis, in the course of fevers; and in anæmia of high degree. It is heard loudest over the right and left ventricles. Potain thinks it is due to tension communicated to the wall of the ventricle by the entrance of blood into its cavity, and is more marked when the wall is least distensible, as is possible when the tone of a muscle is exhausted. This triple rhythm is of bad prognostic omen in chronic Bright's disease.

Reduplication of the Sounds. Reduplication, or apparent doubling, of the heart sounds occurs in various forms. In health the systolic sounds are created at the same time, or synchronously; the diastolic sounds also correspond in time. In so-called reduplication one systolic sound may follow the other, or the aortic and pulmonary diastolic sounds may be created at distinct intervals. As has been stated, in galloping rhythm the idea of reduplication is sometimes transmitted to the ear. Reduplication may take place in *health* under the influence of respiratory movements. The systolic sounds may be doubled at the end of expiration and the commencement of inspiration, while the diastolic sounds are doubled at the end of inspiration and the commencement of expiration. In mitral disease reduplication, or want of synchronous closure of the two valves, is of frequent occurrence. The second sounds are doubled and heard over the base of the heart. Reduplication of the systolic sounds occurs in chronic Bright's disease.

Reduplication, or Doubling of the Systolic Sounds, is heard over the apex or the right ventricle. Several explanations have been given for the cause of the reduplication. At first it was thought to be due to want of synchronism in the action of the ventricles—that one ventricle contracted before the other, due to the fact, of course, that the presence of blood stimulates one but not the other. By Hayden it was thought that reduplication of the first sound was due to the two major elements of the sound acting asynchronously, the muscular sound taking place before the sound produced by the tension of the valves. Dr. George Johnson took the view that the reduplication was due to the contraction of the auricle and ventricle; that the sound produced by the former was heard on account of hypertrophy of the auricle, and heard first because of the natural order of precedence. Thus far the reasons for each view have not been fully established.

Sansom believes that reduplication of the first sound is due to the shock communicated to the contents of the ventricle just before systole, that is, during the auricular-systolic period; in other words, it is due to the indirect effect of the auricular systole. The contraction of the

auricle makes tense the auriculo-ventricular valve of the left side. If it occurs late in the diastole, or just before the systole, reduplication of the first sound is caused; if early in the diastole, reduplication of the second sound is created.

Reduplication of the Diastolic, or Second Sounds. While held by some authorities to occur in a large proportion of healthy individuals at the end of inspiration and the commencement of expiration, other observers, equally careful, think that it is extremely rare. It is of frequent occurrence in the patients of the Philadelphia Hospital. This is no doubt due to the fact that so many of the inmates are the subjects of all forms of lung disease, or disease of the vascular system, with muscular degeneration of the heart, that the equability of the pulmonic circulation is disturbed. There is no doubt that it can be modified or induced by respiration. It is usually heard at the end of inspiration and commencement of expiration. Actual reduplication of the second sound occurs when the normal asynchronism of the closure of the aortic and pulmonary valves is exaggerated. It has been found that the valves of the pulmonary artery close a fraction of a second after the aortic valves. The ear usually fails to appreciate the difference unless there are differences of blood-pressure; when doubled, and appreciated, therefore, it is indicative of a difference in blood-pressure between the two sides of the circulation. Increased resistance in either will lead to increased tension and quickened closure of the valve. The conditions that are associated with the doubling of the second sound are (1) and most frequently, mitral stenosis; (2) obstruction of the circulation in the lungs—tuberculosis, emphysema, and broncho-pneumonia; (3) dilatation of the right ventricle; (4) myocarditis. The sound is heard at the second and third costal cartilages along the left edge of the sternum. It is frequently heard at the fourth and fifth cartilages at the left side. In cases of mitral stenosis it is heard nearest the apex.

Simulated doubling is a sound produced at the mitral orifice. It is difficult to tell it from true doubling or reduplication. It is most distinct at the base of the heart along the left edge of the sternum. Occasionally it is more distinct near the apex than elsewhere. It occurs with the conditions found in true doubling. *Cause.* Sansom, Cheadle and others distinctly point out that the double second sound is of frequent occurrence, and that it is heard most frequently at the apex. Sansom thinks that the cause for simulated doubling of the second sound is the same as for doubling of the first. There is, first, the normal second sound; second, a tension of the mitral curtain producing the second simulated sound. This tension is due to the shock of the blood coming from the auricle to the ventricle.

Abnormal Sounds. The student has observed the character of the sounds and their rhythm. Abnormal sounds may be heard in addition to the normal sounds, or replacing them. These sounds are generated in the pericardium or in the heart itself.

Abnormal Sounds in the Pericardium. They are known as *friction sounds.* They occur in the first stage of pericarditis, and are due to the rubbing of the inflamed surfaces together, either the con-

gested, vascular pericardium, or the membrane covered by lymph. The pericardial friction is usually of a to-and-fro character, and can be recognized as distinct from the heart sounds. It does not necessarily occur with each sound. It is a to-and-fro, systolic and diastolic sound. It may, however, be only systolic. It is heard over the body of the heart, usually in the third and fourth interspaces, or over the right ventricle. It is not transmitted away from the heart. It may be modified by pressure or influenced by the position of the patient. It may disappear entirely with change in position. The idea of nearness to the ear is given by the sound observed in the first stage of pericarditis. It may disappear during the period of effusion, to return after that is absorbed. It must be distinguished from the *pleural friction.* If the patient is asked to hold the breath, the latter will disappear. The pericardial friction is of cardiac rhythm, the pleural friction of respiratory rhythm. It must also be distinguished from the so-called exocardial friction sounds. The pleura adjacent to the pericardium may be inflamed. With each beat of the heart the rough surfaces of the pleura are agitated and generate a friction. It is seated along the edges of the right auricle or left ventricle. It is systolic in rhythm, but has the special characteristic that it is modified by respiration. It may be arrested if the patient holds his breath. It is increased by inspiration or diminished in expiration when the lung recedes from the heart in expiration. The pericardial friction must be distinguished from the crepitations and râles of cardiac rhythm produced by the impact of the heart against the lung. The distinctions between the pericardial friction and murmurs of the heart will be considered later.

Abnormal Sounds within the Heart. They are known as *murmurs*, and may be due (1) to disease of the valve leaflets; (2) to imperfect coaptation of the valves; (3) to change in the character of the blood.

Murmurs due to Valvular Disease. Any valve may be the seat of disease, causing interference with the flow of blood through the orifices. Either there is obstruction to the onward flow of blood through the orifice, or a return, or regurgitation, of blood backward because the valves cannot close properly. In either instance vibrations are produced, which, transmitted to the ear, constitute a murmur. On the one hand, a portion of a valve may be thrown into vibration by the current or by the obstructions of a cusp. The blood is thrown into eddies or vibrations. On the other hand vibration of the particles of blood is created when it is forced through a narrow orifice into a channel beyond of larger calibre. The act results in the production of what is known in physics as a fluid vein. The generation of the vein produces sound. As transmitted to the ear, the sound gives one the idea of rhythmical vibrations, and it is therefore in a measure a musical sound. Distinction from normal sounds: The character of the sound makes it possible to distinguish the murmurs from the normal sounds. The normal sounds are sounds of tension; they are noises, not rhythmical vibration. *Murmurs* are soft and blowing, so-called bellows sounds, or musical. They may, on the other hand, be harsh and rough, varying in degree, compared to the sounds of sawing or filing.

Diagnosis of Different Murmurs. The student has learned that an abnormal sound or a murmur is present. He proceeds further, with two objects in view: first, to determine the orifice at which it is created by the position or seat of the murmur; and second, to ascertain the nature of the lesion at the orifice, on account of which it is produced.

The Seat of the Murmur. We are enabled accurately to determine the seat of the murmur, first, by noting its position of maximum intensity; and second, the direction in which the murmur is transmitted.

The Position of Maximum Intensity. The particular orifice at which a murmur is created has a point of maximum intensity at which the murmur is heard loudest, corresponding with the area at which the normal sound of the respective valve is heard loudest. It may be remembered that the orifices are closely situated, and that, therefore, the murmurs must be generated within a small area, so small that it would be impossible to ascertain at which valve the murmur is created. By the laws of conduction of sound—hence by the influence of the solid heart upon the sounds—the murmurs are conducted away from the point of creation to stations at each of which the respective valve sound is heard with greatest intensity.

1. *Murmurs at the Apex—the Mitral Area.* The murmur is heard loudest, or with the greatest intensity, at the apex. It is due to disease of the mitral valve, because the left ventricle is nearest the chest wall at this point. The solid muscle of the ventricle conducts the sound generated at its valve.

2. *Murmurs at the Xiphoid Cartilage—the Tricuspid Area.* The murmur is heard loudest at the xiphoid cartilage. It is due to regurgitation at the tricuspid orifice, and is heard most distinctly over the lower portion of the sternum, and along the left edge, because the right ventricle is in apposition with the chest wall at this point.

3. *Murmurs at the Second Costal Cartilage or Second Interspace on the Right—the Aortic Area.* When the murmur is heard with greatest intensity at this point it is due to disease of the aortic valves, because the murmur generated at the aortic orifice is conducted to this region by the aorta, which comes nearest to the surface of the chest at this point.

4. *Murmurs in the Second Left Interspace—the Pulmonic Area.* A murmur heard loudest at the second interspace along the left edge of the sternum is generated at the pulmonary valve; it is heard loudest in this area because the pulmonary artery is nearest the chest at this point.

The Direction of Transmission. This will be considered later, although it may be said murmurs due to disease of the aortic valve are transmitted upward from the base; murmurs due to disease of the mitral valve are transmitted away from the apex and toward the axilla.

Having determined the point of maximum intensity of the murmur, hence the valve which is the seat of disease, we next wish to determine the nature of the lesion on account of which the murmur is created. The physical conditions which produce murmurs are present both during the time when the valves should be closed and also at the time when the valves are open and the blood is flowing through the orifices. A murmur which is produced when the valves should be closed, permitting blood to flow through an orifice, is known as the murmur of

regurgitation. A murmur that occurs at the time the blood should in health be passing through an orifice is known as a murmur of obstruction. We have to determine whether the murmur at an orifice is due to regurgitation or whether it is due to obstruction. This is ascertained by the time of the murmur and by the direction in which it is transmitted.

The Time of the Murmur. *Murmurs in the Mitral Area.* The murmur is heard loudest at the apex. It occurs with the *systole.*

1. In health, during this time, the auriculo-ventricular valve is closed. If a murmur replaces the systolic sound there is such disease as to permit of a backward flow of blood, or regurgitation, into the auricle. It is the murmur of *mitral regurgitation.* It is a *systolic murmur.*

2. It occurs before the systole, or during the latter part of the *diastole.* During this time, in health, the blood is flowing through the left auricle to the left ventricle. There must be such disease as to cause obstruction to the flow of blood. It is the murmur of *mitral obstruction.* It is a *presystolic murmur.*

Murmurs in the Tricuspid Area. The murmur is heard at the xiphoid cartilage. 1. It is *systolic* in time. For the same reason as on the left side, the murmur is due to disease which permits of regurgitation, *tricuspid regurgitation.*

2. In rare instances a murmur may be heard in the tricuspid area in the *diastole,* due to *tricuspid obstruction.* It is so rare, however, that it does not need further consideration.

Murmurs in the Aortic Area. The murmur is heard loudest at the second costal cartilage on the right. 1. It is heard with the *systole.* During this time the blood is flowing from the ventricle into the aorta. There is such disease as to cause obstruction at the orifice. It is the murmur of *aortic obstruction.* It is a *systolic murmur.*

2. It occurs with the second sound. During this time, in health, the blood falls back on the aortic leaflets. If they are diseased in such a degree as to permit a portion of the blood to flow backward into the ventricle, a murmur is created. Regurgitation is produced and a murmur is heard—the murmur of *aortic regurgitation.* It is a *diastolic murmur.*

Murmurs in the Pulmonary Area. 1. It occurs with the systole. The murmur is heard loudest at the second interspace on the left. The pulmonary orifice is affected in a similar manner as the aortic orifice under the same circumstances. The murmur is due to *pulmonary obstruction.* It is exceedingly rare.

2. It occurs with the *diastole,* for the same cause as in aortic regurgitation. It is of such extreme rarity it can practically be excluded. It is due to *pulmonary regurgitation.*

Murmurs are divided as to time into systolic and diastolic murmurs. The above shows that we may have practically only three systolic and two diastolic murmurs. The systolic murmurs are aortic obstruction and mitral and tricuspid regurgitation. The diastolic murmurs are aortic regurgitation and mitral obstruction; the former occurs in the first part of the diastole and represses the second sound; the latter

in the diastole, either in the last part or before the systole, or in the midst of the diastole.

Direction in which the Murmur is Transmitted. This depends upon the situation of the murmur and the time at which it is produced.

Murmurs in the Mitral Area. A murmur which is produced at the apex with the systole, caused by regurgitation at the mitral orifice, is transmitted into the axilla and may be heard at the angle of the scapula. The murmur which is produced in the same area before the systole is not transmitted over the body of the heart. It is heard at the apex, or a little inside of the apex, or may rarely have its point of maximum intensity in the third interspace.

Murmurs in the Tricuspid Area. The murmur of tricuspid regurgitation is not transmitted. It is heard over a relatively large area, depending upon the degree of loudness of the sounds.

Murmurs in the Aortic Area. The murmur, systolic in time, heard at the second costal cartilage on the right, due to aortic obstruction, is transmitted in the direction of the blood current. The sound is conducted by the vessels and by the fluid; it is therefore heard along the course of the aorta and in the carotid arteries. The murmur of aortic regurgitation, heard in the same area, is transmitted downward along the course of the sternum. It may be transmitted to the apex, or may be along the sternum alone. The left ventricle conducts this murmur.

Character of Murmurs. Murmurs are studied in accordance with the above, as to their situation, their time, and the direction in which they are transmitted. In addition, we study the *character* of the murmur and the degree of *loudness*. By the character of the murmur we are aided (1) in distinguishing them from heart sounds; (2) in estimating the nature of the lesion that produces the murmur; (3) in judging, in the case of murmur of mitral obstruction, of the presence or absence of that disease.

From Normal Sounds. Normal sounds are sounds of tension; murmurs are sounds of vibration. The normal sounds of the heart have been recognized by syllables "*ubb*," "*dupp*," "*od*," and abnormal sounds of endocardial origin by "*uf*," "*uv*," "*us*," "*ush*," or by full vowel sounds as "*oo*," "*u*," "*ah*," and "*aw*," by musical tones, or by interrupted tones, or hearing general sounds as "*urr*" or "*orr*." *The nature of the lesion.* The murmurs may be rough or rasping, musical or whistling in character. They may be high in pitch or low in pitch. Murmurs that are rough and high in pitch, are usually due to disease of the valves which is caused by thickening or stiffening of the leaflets, or to the projection of an atheromatous plate into the lumen of the orifice. Such conditions occur in chronic endarteritis and chronic endocarditis or valvulitis. On the other hand, murmurs that are soft and low in pitch are usually due to a physical condition which causes swelling of the valve or occlusion by soft exudations; they are heard in endocarditis of rheumatic origin, or the malignant form of endocarditis. The only murmur which has special characteristics is the murmur of *mitral obstruction.* It is a prolonged murmur of a churning or grinding character, sometimes rippling, and from which we get the idea

that fluid is being forced through a narrow channel. It is usually presystolic, but may occur in the middle of the diastole. *Loudness.* The *loudness* of the murmur is not of special significance, although, in general, it may be said that it indicates good compensation, and that the force which generates the murmur is sufficient to meet the demands of the circulation. Loud murmurs may become weak, and this change in character of the sound is of serious omen.

Change of Murmur. The student will often find that after a patient has been under treatment for a short time the murmurs disappear. This is probably due to the fact that there is complete compensation. On the other hand, it may be necessary to bring out a faint murmur or increase its intensity by having the patient move about; this renders it more distinct by inducing more rapid action of the heart.

Murmurs due to Incompetency. The valves are sometimes unable to close properly. The cavity of the ventricles may increase in size, so that the valves do not coaptate properly to close the widened orifice. The tricuspid and mitral valve leaflets are often thus made incompetent. Mitral and tricuspid regurgitation ensue. The murmurs are soft and low in pitch and not widely transmitted; the heart is dilated.

The Murmurs of Anæmia. Having ascertained a murmur and the orifice at which it is created, we have to distinguish whether the murmur is due to disease of the valves or whether it is due to anæmia. The murmurs of anæmia have some characteristics which aid in distinguishing them from the true organic murmurs. The most important of these are: (1) the situation of the murmur; (2) its character; (3) the direction in which it is transmitted; (4) the time; (5) the associate conditions. The murmurs of anæmia may be heard at any orifice, but are usually heard at the second costal cartilage, or the third interspace, on the left side. They are generated at the pulmonary orifice, or in the cone of the right ventricle. They are soft in character, and low in pitch. They are systolic in time and are not transmitted away from the heart. The murmur at the pulmonary orifice may be heard as high as the second interspace, but otherwise is not transmitted. Murmurs of anæmia are also heard at the apex, at the aortic cartilage, and over the tricuspid area. They are comparatively infrequent in these situations, but partake of the same nature as the murmur heard at the pulmonary orifice. The heart does not undergo hypertrophy of special portions. Dilatation, fatty degeneration or hypertrophy may be present. We distinguish the murmur of anæmia, in addition, by its association with murmurs in other parts of the vascular system. The murmur in the jugular veins is usually associated with an anæmic murmur heard over the heart. Its characteristics and mode of distinction have been described elsewhere.

The Significance of Murmurs. Murmurs that are heard at the various orifices indicate disease at the orifices causing obstruction or incompetency of the valve, or disease of the blood, or disease of the vessels in intimate relation with the heart. The systolic murmur at the second costal cartilage on the right may be heard in structural disease at the aortic orifice, causing obstruction or atheroma of the aorta, or in cases of aneurism just above the valves, or of anæmia, or chlorosis, and in some affections

with vasomotor neuroses, as Graves' disease. Before concluding that the murmur is due to disease of the valves we must decide the absence of the other three conditions. *Atheroma of the aorta* is most difficult to distinguish, because the character of the murmur is the same and the associated conditions are similar. In both there may be a previous history of gout, rheumatism, syphilis, or of alcoholism. The latter is associated with atheroma in other arteries of the body, and with degenerative changes that accompany atheroma. In young subjects, in whom there has been a direct history of rheumatism, or when the process has followed septicæmia, the probabilities are, in nearly all the cases, that the murmur is due to aortic obstruction. To distinguish the murmur of anæmia, chlorosis, or Graves' disease is often difficult. The associate symptoms are different, and then the changes in the blood are such as to indicate the nature of the murmur.

Secondary Effect of Valve Lesions on the Heart and Pulse. While we are enabled by the time of the murmur, the position, and the direction of transmission, to affirm the nature of the disease at respective valve orifices, other physical signs of diagnostic significance aid us in determining more precisely the lesion and its seat. They depend upon the secondary effects of the lesion upon the heart and upon the circulation. In aortic obstruction, on account of obstruction to the flow of blood, the left ventricle hypertrophies; moreover, the blood stream is lessened in volume, and hence the pulse is small and of high tension. The physical signs of hypertrophy and small pulse are corroborative evidence of this lesion at the left orifice. In aortic regurgitation the blood flows back into the ventricles. On this account, therefore, some dilatation takes place, a dilatation which, if compensation is perfect, is overcome by hypertrophy. The signs, however, of enlarged left heart are present, as indicated by inspection, palpation, and percussion. But the pulse of aortic regurgitation is of the greatest diagnostic significance. With the finger on the radial, the impression is at once received of recedence of the pulse wave as soon as it strikes the finger. This is more marked if the hand is elevated. It is the water-hammer, or Corrigan's, pulse. In mitral regurgitation, the left auricle does not change, but the stress is thrown upon the right side of the heart, and we have the signs of right-sided hypertrophy and dilatation; but more marked than this is the evidence of tension of the pulmonary artery, which is shown by accentuation of the second sound (see p. 358). In mitral regurgitation, the blood flows back into the auricle and engorges the venous system. The arterial system is devoid of blood, and hence the artery is emptied. The pulse is small and feeble; the coronary arteries are not fully supplied with blood, in consequence of which there is a diminished amount of blood to nourish the ventricles. Dilatation or failure in nutrition soon ensues, and the heart is unable to do the work expected of it. In addition to the small and feeble pulse, there are inefficient and hurried contractions, on account of which the pulse is irregular and intermittent.

In mitral obstruction, in addition to the characteristic murmur, the thrill is of great significance. Moreover, the left auricle hypertrophies, and shortly afterward the right heart. It is accompanied by an accentu-

ated pulmonary second sound, and frequently by doubling of that sound. The pulse is small and feeble.

Examination of the Arteries and Veins. The state of the circulation in the arteries and veins is greatly influenced by the condition of the heart. Examination of them yields data of diagnostic value in the discrimination of heart disease. It is appropriate that such examination should be considered before proceeding with the diseases of the heart.

The Arteries. Inspection. By inspection pulsation may be observed or any undue swelling or change in the course of the vessels. With the exception of pulsation in the carotids, which may temporarily increase under excitement, pulsation of the vessels is not usually seen in health. The arteries open for inspection are, in old people, the aorta rarely at the episternal notch, the temporal, the innominate, the carotids, the subclavian, the brachial and radial arteries, the abdominal aorta in thin subjects, the femoral arteries and the posterior tibials.

THE ARTERIES IN THE NECK. Temporary pulsation of these arteries has been spoken of. This occurs in excitement. It is commonly seen in anæmia. The throbbing is marked in exophthalmic goitre. It is striking in aortic regurgitation. It often attends the vascular changes of old age. It may be due to atheroma or aneurism. It is always suggestive of aortic valvular disease. The innominate artery often visibly pulsates in the neck, and has been observed to be so large as to simulate aneurism. The youthful age of the patient points to throbbing of neurosal or hæmic origin. The subclavians may pulsate for the reasons above mentioned. They may also be seen to pulsate if the lungs are consolidated or shrunken by disease.

THE AORTA. Pulsation of the thoracic aorta is determined by the occurrence of an impulse in the course of the vessel. The position of this impulse will be described under the head of aneurism. Pulsations in the course of the aorta are not always due to disease of the vessels. The aorta may be pushed against the chest wall, or the lung structure which overlaps it normally may be withdrawn by shrinkage.

The Abdominal Aorta. Pulsation of the abdominal aorta is often the cause of serious distress and complaint. The violent throbbing keeps the patient awake at night, and renders his previously nervous state more nervous and irritable. The pulsation is usually seen in the epigastrium. It is more frequent when the vessel is not diseased, in neurasthenic subjects. It occurs reflexly in patients with dyspepsia or organic disease in the upper abdominal tract. The shock of the pulsation is transmitted to the hand with considerable violence. The impulse is diffused, but not expansile.

Epigastric pulsation is also due to the transmission of the impulse of the aorta by enlargement of the pancreas or tumors of the stomach or the omentum. The transmitted pulsation is distinct. It is believed to be present when the tumor can be defined and when a sensation of lifting is transmitted to the hand. The physical signs of aneurism are absent. If the patient lies on the abdomen, or in the knee-chest position, the tumor falls away from the aorta, and the impulse is not readily

transmitted. Epigastric pulsation is also due to aneurism of the abdominal aorta. The pulsation is distensile or expansile, and the aneurismal sac can be defined at times. The other physical signs of aneurism are present usually, namely, thrill, dulness over the tumor, a murmur on auscultation. Often in the above conditions reliance cannot be placed on the physical signs alone. The history of the subjective symptoms of disease of other structures must be carefully inquired into. Aneurism rarely occurs without some evidence of arterial sclerosis or some physical effect upon the circulation in the bloodvessels. Accentuation of aortic second sound on the one hand, variations in the femoral pulse, high arterial tension and the evidences of sclerosis, favor aneurism. While epigastric pulsation due to pulsation of the aorta usually occurs in neurotic subjects, and hence in the earlier periods of life, yet such pulsation is frequently seen in the aged, and, with fibrous thickening about the pylorus, or contraction of the omentum, it may easily be confounded with malignant disease, which is also more common during this period of life. Cancer of the stomach has been diagnosticated under these circumstances when the pulsation was simply reflex from chronic gastritis. Some time ago a private patient in the Presbyterian Hospital had extreme pulsation of the abdominal aorta with great local discomfort on account of the throbbing. She was sixty-five years of age, and had within the past two years nursed her son through tuberculosis. She failed in health, and came to the hospital emaciated, with pronounced chronic gastritis and diarrhœa. On examination, above the umbilicus a distinct tumor was felt, which she had been told was due to carcinoma. It was hard and painless; the physical signs of aneurism were not present; the pulsation was extreme. A second tumor, not so large, was felt in the right hypochondriac region. Both tumors were dull upon percussion and surrounded by tympanitic areas. They were also movable. While it is impossible to state the nature of the tumors, it seemed to me they were tuberculous, or simply fibrous, and would not influence the patient's immediate welfare. Under treatment, the pulsation disappeared; the gastro-intestinal symptoms were relieved entirely; the patient rapidly gained in weight and strength; the tumors continued, but they are not so distinctly outlined because the previously scaphoid abdomen has become distended (six months under observation). The questions arose for decision: Was the epigastric pulsation due to a throbbing aorta or transmitted by an obscurely defined mass in that region? Were the other tumors secondary carcinomatous nodules? The diagnosis must be made by attention to all concomitant circumstances and phenomena that surround cancer. (See Symptomatology of Morbid Processes.) *Fœcal accumulations* in the colon may be made to pulsate by the impulse of the aorta and cause exaggerated epigastric impulse. Evacuation of the bowels must be secured before definite conclusions are arrived at.

Epigastric impulses due to the above-mentioned causes must not be confounded with the impulse in the same situation due to hypertrophy of the right ventricle or to the shock of the hypertrophied heart transmitted to the left lobe of the liver. In hypertrophy of the right ventricle or dislocation of the heart from disease within the chest, the

impulse may be seen to the right or left of the xiphoid cartilage. The symptoms and signs of right-ventricle hypertrophy explain the pulsation.

The Smaller Arteries. By inspection of the arteries beyond the abdominal aorta we are able more distinctly to recognize frequently the condition known as arterio-sclerosis. Similar examination of the brachial and radial arteries reveals the same condition the changes of which are spoken of when that disease is considered. (See Arterio-sclerosis.) But pulsation of the above-mentioned peripheral arteries may be due to other causes. In hypertrophy of the left ventricle arterial pulsation is prominent, although more marked in the vessels near the heart, as the carotids. In insufficiency or regurgitation at the aortic orifice pulsation is also frequently seen.

Elongation of this artery, so that instead of a straight tube, it becomes a sinuous canal, turning and twisting at short intervals, is seen in endarteritis.

Capillary Pulse. The capillary pulse is seen under the finger-nails or in the skin after hyperæmia is induced by the observer firmly stroking the skin with his nail. It may be seen inside the lips, if they are pressed upon by a piece of glass. There is rhythmical pulsation of the capillaries, from which the surface becomes alternately white and red. It is a sign of aortic insufficiency.

Palpation. Reference must be made to the sections on aneurism, arterio-sclerosis, and the pulse. The results of inspection are confirmed. In addition, the artery is examined to determine its tension, the character of the coats, and the presence of thrills. *Pulsation of Organs.* It is said that in aortic regurgitation an arterial liver pulse, similar to the venous liver pulse can be felt when the hands are placed over that organ. Similar pulsation may be felt in the spleen.

In examining the arteries it is important, as has been detailed in the pulse, to compare the arteries of the two sides. Often the pulse wave in them is found to be unequal in force, in volume, and in time. This is almost always due to obstruction to the passage of the blood. When not due to *endarteritis* or to *aneurism*, it is due to pressure of a tumor on the vessel somewhere in its course. A thrombus or embolus in the artery may likewise cause the condition. A difference in the radial and the femoral pulse points to obstruction in the thoracic or abdominal aorta. Anatomical variations must be remembered.

The Pulse.

The pulse is an index of the force and rhythm of the heart's action and of the state of pressure, or tension, which it maintains in the arteries.

General Considerations. The *frequency* of the pulse before birth is from 120 to 140 beats in the minute. From this time it diminishes in frequency up to adult life, 72 being then accepted as an average; the number of beats, however, is often under 72, and sometimes over that. In old age the pulse rate is again increased. Sex has

some influence. The rate is slightly higher in females than in males of the same age.

The frequency of the pulse is subject to diurnal variations, at times corresponding with the diurnal rise and fall of temperature. The rate will therefore be highest in the afternoon and evening and lowest in the early morning hours.

The position of the body has also a modifying influence. The pulse is more frequent when a person is standing than when he is sitting, and more frequent when he is sitting than when he is lying down. Walking, running, bodily and mental exertion, fear, and excitement all tend to accelerate the pulse.

During and for one or two hours after a meal the pulse rate is higher, especially if an alcoholic or other stimulant, such as coffee, has been taken.

How to Take the Pulse. To make a correct count of the frequency of the pulse, the conditions just mentioned, as normally modifying its rate, should be borne in mind. If the object of the count is to determine the rate which is normal for a particular individual, several counts will be necessary at different times and under different conditions, such as sitting and standing. The best time for the physician to take the pulse will have to be determined by his own judgment in each case. If the patient comes to his office and is excited by the prospect of an examination, it will be well to wait until he becomes calm. On the other hand, if he is calm at first, a count at that time is to be preferred to one made after the patient has been disturbed by a physical examination. In the same manner, on visiting a patient at his house, the judgment of the physician must decide whether to count the pulse immediately on his arrival or to postpone it until, by general conversation, all apprehension and alarm on the part of the patient have been allayed. In general it may be said that if the physician finds, upon his arrival, that the pulse is more frequent than the condition of the patient would lead him to expect, he should wait a while, endeavor to find out whether anything has served temporarily to disturb the circulation, and then make the count when the conditions are most favorable. Some patients are so nervous that the mere act of placing the finger upon the wrist sends the pulse rate up ten or twenty beats in the minute. In such cases the effort should be made to obtain a count without the patient's knowledge by observing the pulsations of the temporal or carotid. In other cases it may be well to entrust the counting of the pulse to the nurse or a member of the family. In infants and young children, count while they are asleep. In febrile conditions the count is more likely to be too high than too low.

In hospital practice, or when a nurse is in constant attendance, the pulse and respiration should be taken at the same times as the temperature. But the nurse must be warned against taking them under dissimilar conditions upon successive days. For example, the pulse should not be taken one day while the patient is lying down, quiet and comfortable, and compared with the count the next day when the patient is sitting up or has just had some hot liquids, or a spell of coughing, or been subjected to some other disturbing influence.

The preferable position is the recumbent one in the case of patients in bed, and the sitting position in those not confined to bed. Care should be exercised in all cases to see that the patient's position is easy and comfortable and that nothing obstructs the artery or interferes with the unimpaired flow of the blood.

The wrist is the place usually selected at which to feel the pulse At this point the radial artery passes over the radius, and can be readily compressed and its character made out. An old-fashioned rule prescribes that three fingers should be applied to the artery, the index finger of the physician being nearest the heart. In particular cases it may be advisable to count the pulse at the temporal or carotid artery. The fingers should be applied so that the beats can be most distinctly felt. The beats are counted for fifteen seconds by the second hand of a watch when only an approximately correct count is desired, or when time is a factor, and then multiplied by four. It is better to count the pulse for half a minute, and still better for a full minute.

The arteries of the two sides must be compared. Difference in the force, volume, and time may be due to the anomalous distribution of arteries. In disease, it may occur in aneurism and atheroma, in pressure on the trunk from external disease, and in embolism and thrombosis.

Condition of the Walls of the Artery. The condition of the artery is often of more importance than the pulse rate. A healthy radial artery in a person not advanced in years can be compressed easily against the radius without the finger being able to differentiate the artery from the other tissues. But as age advances, and as the result of certain constitutional diseases—syphilis, gout, chronic endarteritis, alcoholism, and others—the artery tends to become thicker, so that in pronounced cases it cannot be obliterated, but is rolled like a cord or pipe-stem between the compressing fingers and the bone. The small specks or plates of atheroma, feeling like hard particles in the coats of the artery, may be felt. The artery has a beaded feeling. Fatty degeneration of the organs is likely to occur when the arteries are in this condition, and apoplexy is to be feared.

Tension. Tension is the word used to express the degree of blood pressure—that is, of distention of the arteries. Normally, the pulse nearly or quite subsides between the beats, but little pressure being required to obliterate it. *High tension* may be said to exist when the artery remains continuously full between the beats (Broadbent). It is produced by plethora; increased heart action; contraction of the arterioles, as by chill; and obstruction in the capillaries. The conditions which bring about obstruction in the capillaries in the order in which they are enumerated by Broadbent are: (1.) Age. The liability to high arterial tension increases with the age, especially after middle-life. (2.) Heredity. There is in some families a marked tendency to high tension. The younger members show its effects in headaches and bilious attacks, while the older ones develop chronic heart disease and apoplexy. (3.) Disease of the kidney. Parenchymatous, but especially interstitial nephritis, is associated with high arterial tension; this, with accentuation of the aortic second sound, is one of the early and, therefore, one of the most valuable indications of chronic Bright's disease.

(4.) Gout. Gout and lithæmia are almost always accompanied by high arterial tension. (5.) Diabetes in old persons associated with gout. (6.) Lead-poisoning. (7.) Pregnancy. (8.) Anæmia. (9.) Emphysema and chronic bronchitis. (10.) Mitral stenosis.

As regards arterial tension in persons presenting signs of angina pectoris, Sansom asserts that if the tension is increased, even though the signs are not typical, the fear, present or remote, of true angina is justified. On the other hand, if there is persistent low tension, especially during the painful crisis, it is almost certain the affection is a false angina.

Low tension of the pulse is characterized by a softness and a compressibility in excess of the normal. This, like the high-tension pulse, may be a family peculiarity. It is met with in conditions of great depression and exhaustion and wherever there is marked cardiac weakness. It is most common in fevers, particularly in typhoid, in which also an accompaniment of low-tension pulse, namely, dicrotism, is met with in a marked degree. Fat persons are apt to have low-tension pulses, and it may occur in any person temporarily under the influence of external warmth and moisture, such as a hot bath, or after taking hot drinks, or under the influence of depressing emotions, diarrhœa, or sudden copious urination.

Volume. The volume of the pulse should be noted. It is usually large in conditions of pyrexia and when the tension is low. A small pulse is met with in many conditions other than weakness of the heart muscle. In aortic stenosis the pulse is small, and in mitral stenosis it is small, of high tension, and frequently irregular. In general contraction of the arterioles, as happens under the influence of a chill, the pulse is small. In Bright's disease it is sometimes very small, slow, and hard. Some care will be required to differentiate such a pulse from a weak pulse. In acute peritonitis the pulse is apt to be small and hard.

Rhythm. The rhythm of the pulse is of diagnostic importance. In health one beat succeeds another at equal intervals of time, and the successive beats are of the same force and quality. Here also, however, as in other conditions, there are variations within physiological limit. In some persons the pulse rate is somewhat accelerated during respiration and becomes slowed in the pauses which follow breathing.

In disease, disturbance of the rhythm occurs as intermission or as irregularity. *Intermission* signifies a stopping of a pulse beat; several normal pulse beats succeed each other, and then the pulse is absent during the time occupied by one or two beats. The intermission may occur at regular or at irregular intervals—that is to say, every third, fifth, or sixth beat may be wanting, or the intermission may be irregular—now a second, the next time a fifth, or a third beat being absent. Moreover, the intermittent pulse may be constant, or it may, and more frequently is, only occasional. It is not characteristic of any one disease or condition, and it may exist without the patient's knowledge and without producing any perceptible effect upon his health. Sometimes it is met with in a fatty heart, and this disease may be suspected if the intermittent pulse is associated with a weak first sound of the heart without valvular lesion, and evidences of failing circulation, such as œdema of

the feet. More frequently, however, the intermittency is a symptom of nervous depression, or is caused by tea, coffee, tobacco, or digitalis. So far as prognosis is concerned, it is much less serious than irregularity. Broadbent says he has met with it at the age of eighty, when it has been known to exist for forty years.

Irregularity is characterized by differences in time, force, or volume of successive beats. A full beat is succeeded by another, which is smaller and weaker, or successive beats occur at irregular intervals of time. Irregularity may or may not be associated with intermission. In advanced cases of mitral stenosis the pulse is both irregular and intermittent. The irregularity may be habitual or occasional; the former is due most frequently to mitral lesions, but sometimes occurs without assignable cause, and is attributed to disturbance of the nerve supply; the latter is due to digestive disturbances and to the effect of nicotine and digitalis. Irregularity is not incompatible with health, but is much more likely to be of serious import than intermission. It occurs in diseases of the brain, in degeneration of the heart as well as in valvular lesions, and in grave cases of febrile diseases, such as typhus and typhoid, when the heart muscle is involved. Some cases of Graves' disease are characterized by great irregularity instead of excessive rapidity of the pulse. Irregularity may occur in rheumatoid arthritis also, though increased frequency is the rule.

The Pulse in Diagnosis. The *frequency* of the pulse is of aid in diagnosis. 1. The pulse is *increased* in frequency in all febrile diseases, and generally in the proportion of eight to ten beats for each degree of rise in temperature above 98°. But there are important exceptions. In *typhoid fever* the pulse is slower in proportion to the temperature and the gravity of the disease than in most of the other acute febrile diseases. It may not beat above 85 in mild cases, and in severe cases frequently does not rise above 100. Consequently a pulse of 120 is of much graver import than it would be in other diseases. It may be more frequent during convalescence than during the febrile stage. This pulse rate helps to differentiate it from tuberculosis, malignant endocarditis, and septicæmia.

2. The pulse of *scarlet fever* often aids materially in diagnosis. A pulse of 120 to 160 is the rule from the development of the sore-throat to the completion of the eruption. In measles, rubella, diphtheria, and follicular tonsillitis it is much slower during the early stages.

3. In *Graves' disease* great frequency of the pulse is the essential and the most constant symptom of the disease. The pulse may be constantly considerably over 100, and in attacks of palpitation 200 or more. In these attacks there may or may not be præcordial distress and mental anxiety. Here belong the cases described as paroxysmal hurry of the heart, etc., the thyroid and ophthalmic symptoms being absent.

4. Cases have been reported of extreme frequency of the pulse (160–240) without palpitation, dyspnœa, or any sign of Graves' disease. Some of the patients have been able to perform much bodily and mental labor, notwithstanding that the rate mentioned was maintained persistently for weeks. To this class of cases the name *tachycardia* has been applied until their pathology is understood.

5. *Mitral stenosis* may be latent until great excitement, over-exertion, and particularly running or forced marches bring on palpitation, or simply abnormal and persistent frequency of the heart's action, with or without dyspnœa. In all forms of valvular disease, except aortic stenosis, with failing compensation, in collapse, in weakening of the heart, and in central or peripheral vagus disease, the pulse is increased.

6. Attention has been called, especially by Dr. J. Kent Spender, to acceleration of the pulse as an early symptom of *rheumatoid arthritis*. The pulse increases gradually until it reaches a range of 110–120, and it persists at that rate with little diurnal variation, even after the arthritic symptoms subside.

7. In *locomotor ataxia* permanent moderate acceleration of the pulse (90–100) is a frequent symptom.

8. In *puerperium* increased frequency with irregularity of the pulse is a surer indication of intra-uterine mischief than is the temperature. So, too, in all cases in which there is a focus of suppuration so situated that the pus can be absorbed into the circulation but not discharged externally, the pulse shows by its increased frequency that absorption is going on.

A *slow* pulse (bradycardia), under 60, like a frequent pulse, is sometimes habitual, and sometimes a family characteristic. Pathologically, it is met with in conditions which increase the resistance in the arteries, such as Bright's disease, especially acute glomerulo-nephritis; but it is especially common in jaundice. The bile acids have the effect of slowing the heart.

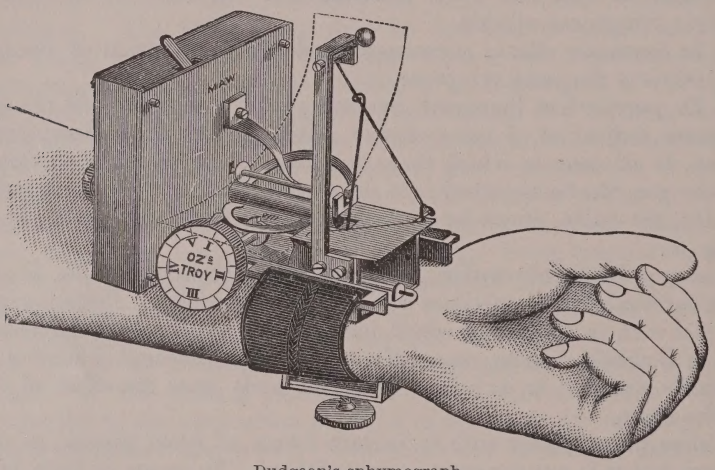
A slow pulse is met with in certain forms of *heart disease*, as aortic stenosis, but it is not constant in any of them. It occurs in fatty degeneration, especially when due to obstruction, by atheroma or otherwise, of the coronary arteries. When it appears in the late stages of valvular affections or specific diseases with cerebral symptoms it is usually a sign of danger. It is seen in articular rheumatism (Atkinson). According to Riegel it is most common in *convalescence from acute disease*, particularly pneumonia, typhoid fever, erysipelas, and rheumatic fever. It is also frequently encountered in diseases of the *digestive organs*, and of the urinary organs, particularly *acute nephritis*. Moreover, it is generally slow in *myxœdema*, and slow and irregular in *epilepsy*. It is slow not uncommonly, also, in *melancholia* and in the early stage of *cerebral meningitis* and in tumors and cerebral hemorrhage.

The Sphygmograph. The sphygmograph, as its name implies, is an instrument for recording in writing the volume, force, frequency, tension, and general characteristics of the pulse. Many forms of the instrument have been devised since the first one of Marey. The later models have the advantage of simplicity and ease of application. One of the most convenient is Dudgeon's. It has its faults, particularly in exaggerating the vibrations when the pulse is large and the heart is acting violently; nevertheless, with care, trustworthy tracings can be obtained in all ordinary cases. No matter what instrument is used, the value of the tracing depends very largely upon the personal skill and experience of the one who takes the tracing; hence the sphygmograph

occupies a position very different from the thermometer and other instruments of precision. While it is true that a person can learn to detect nearly all the variations of the pulse by palpation alone, yet the tracing has the great advantage of permanency, and many persons are led to palpate the pulse more carefully by seeing in a sphygmographic tracing a dicrotism or irregularity which had escaped their attention.

The best sphygmograph for ordinary clinical work is that of Dr. Dudgeon (see Fig. 61). It is very compact and easy of application.

FIG. 61.



Dudgeon's sphymograph.

The expansile pulsation of the artery is communicated by a system of levers to a needle, which graphically records the qualities of the pulse upon smoked paper.

Directions for Using Dudgeon's Sphygmograph.

1. Wind up, by the button, the clockwork contained in the box. The clockwork carries the smoked paper under the writing-needle.

2. See that the patient is in a comfortable position, and have him hold toward you either hand with wrist exposed, fingers gently flexed, and muscles relaxed.

3. Apply the instrument by slipping the band over the hand, the free end of the band being passed through the retaining clamp. The metal box is placed toward the elbow.

4. Now adjust the instrument by placing the bulging-button which connects with the levers directly over the radial artery at its most accessible point.

5. Keep the instrument accurately in place with the left hand, and draw the band through the clamp with the right until the writing-needle plays freely with each pulsation of the radial artery, then fasten the band by screwing up the clamp.

6. Introduce the smoked paper between the rollers and under the writing-needle.

7. Vary the pressure by means of the thumb-screw, which connects with an excentric, until the best apparent amplitude of vibration is obtained.

8. Instruct the patient not to move the fingers or hand, and further steady them for him with your own right hand.

9. Start the clockwork by pushing the bar at the top of the clockwork box.

10. Allow the paper to run through, and then stop the clockwork.

The clockwork is so regulated that five inches of smoked paper pass through in ten seconds, so that six times the number of pulsations recorded on the paper represents the pulse rate per minute. Each instrument, however, should be tested and its time determined. The clockwork should be wound up for every other tracing.

Considerable practice will be required to take a tracing rapidly and accurately, in spite of the simplicity of the mechanism.

Several tracings should be taken at different pressures and compared, or, what is better, as suggested by Sansom, stop the clockwork and alter the pressure two or three times, so as to have the effects of varying pressures on one tracing.

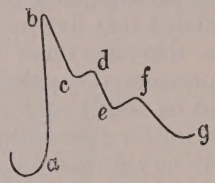
The technique of sphygmography needs a few words. Smoked paper is generally used for the tracings. A paper glazed upon one surface and rough upon the other has some advantages. This paper has to be cut in strips about seven-eighths of an inch wide and six inches or more long. The cutting should be done with care so that the edges are smooth and even, otherwise the paper sticks in the instrument and the tracing is spoiled. The glazed surface is blackened by holding it above the flame of a small piece of burning gum camphor. For convenience a strip of tin, bent upon itself at each end, so as to catch and hold about an inch of the ends of the paper, may be used to prevent the fingers from becoming blackened and to preserve the ends of the paper unblackened for memoranda. The blacking should not be too thick, otherwise the needle will not plough through it easily, and the white line of the tracing will not be distinct. After the tracing has been made, the name of the patient, the diagnosis of his disease, the date of the tracing, and the amount of pressure employed should at once be scratched with a fine-pointed pen upon the blackened surface beneath the tracing, or written in ink upon the unblackened end of the paper. The tracing is then ready for preservation. This is done by dipping it in a solution of shellac or in tincture of benzoin (gum benzoin ℥j, alcohol f℥vj); the alcohol evaporates and leaves a smooth, glazed surface. Dr. Dudgeon recommends as a varnish a solution of gum damar ℥j, rectified benzoline f℥vj. When the tracing is likely to be subjected to friction, a second or third coat should be applied subsequently.

Explanation of the Normal Pulse Tracing.

With each contraction of the left ventricle a volume of blood is forced into the aorta, which distends it, the distending impulse being transmitted wave-like to remote arteries. This distending impulse lifts the button of the lever sharply upward, forming the so-called percus-

sion up-stroke, *a b*. But the distending impulse is exaggerated by the system of levers, and having been thrown up too high the lever falls by its own weight too low, so that it is again caught and lifted by the tidal blood, forming the tidal wave, *c d e*. The gradual descent of the lever is again interrupted at *e f g*, forming a wave, called the dicrotic wave, due to the recoil of the blood from the closure of the aortic valves.

FIG. 62.

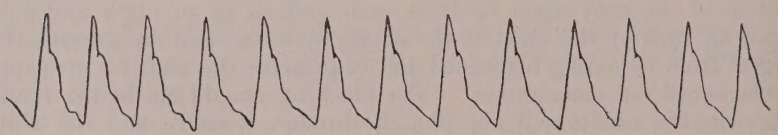


a b, percussion up-stroke; *a b c*, percussion wave; *c d e*, tidal wave; *e f g*, dicrotic wave; *d e f*, aortic notch; *f g*, diastolic period.

INTERPRETATION OF PULSE TRACINGS. Sphygmographic tracings must be interpreted in accordance with the known peculiarities of the patient, his history, and the associated physical signs.

1. *The Amplitude.* The height of the percussion stroke varies considerably in health. It is increased in conditions which bring about low tension and rapid systolic contractions of the heart. Hence the febrile pulse is usually one of considerable amplitude. It is increased also very markedly in aortic regurgitation. Suddenness of systole rather than force determines the height of the up-stroke (see Fig. 63).

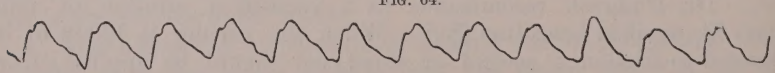
FIG. 63.



Tracing from a case of aortic regurgitation.

2. *Obliquity of the Percussion Stroke.* Normally the percussion stroke ascends vertically from the base line. A tendency for it to incline forward indicates a weak and laboring heart or an aneurism interposed between the radial artery and the heart. In the latter case there is also a tendency to rounding of the summit of the percussion

FIG. 64.



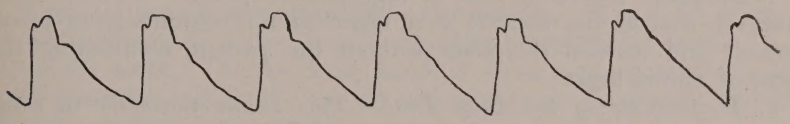
Tracing from a case of aneurism of the aorta.

wave, and the up-stroke is generally short. There is usually also irregularity in successive pulsations, some showing the gradual ascent and rounded summit much better than others. Sometimes, however, when aneurism exists, there is no evidence of it in the tracing, and differences upon the two sides are not always significant (see Fig. 64).

Disease at the aortic orifice and the intervention of a considerable quantity of subcutaneous fat, or of any growth superficial to the vessel, may cause a marked obliquity of the percussion stroke. Sansom asserts that, such causes excluded, as well as aneurism and organic disease of the aorta and its valves, a sloping line of ascent, observed under various gradations of pressure, indicates feebleness of the left ventricle. He considers it of higher diagnostic value than irregularity, which is often neurosal.

3. *Increased Breadth of the Apex of the Percussion Wave.* The breadth of the apex of the percussion wave indicates the time during which the artery is kept full by the systole of the left ventricle. When the left ventricle acts slowly and forcibly the arteries will be kept dis-

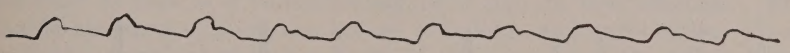
FIG. 65.



From a case of aortic stenosis, showing increased tension and the *pulsus bisferiens.*

tended for a longer time, and this distention will be manifest in broadening of the apex of the tracing (see Fig. 65). The degree of distention of the artery is called the tension, hence a broadening of the apex is an evidence of high tension. As the word high does not indicate the

FIG. 66.



From a case of mitral stenosis, showing increased tension and some irregularity.

duration of the tension, Sansom has very properly suggested that we should speak of persistent high tension as prolonged tension. This, then, is the significance of the broad top of the tracing.

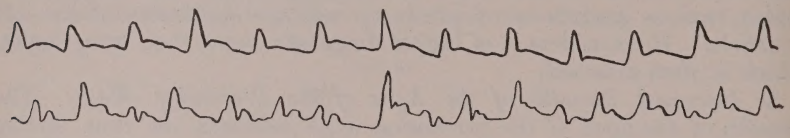
Prolonged arterial tension occurs when there is a strong heart acting slowly, a large volume of blood, or obstruction in the capillary circulation. (For specific causes, see under Tension.)

The amount of pressure required to develop the characteristics of a pulse, and still more, the amount required to obliterate it, is a good index of the degree of tension present. Some pulses, however, appear to the touch to be of prolonged tension, but a sphygmogram does not show it. Such cases are often explained by the fact that the heart has begun to fail under the strain put upon it by prolonged obstruction in the capillaries. There may be regurgitation also from the mitral or aortic orifice.

4. *Acute Angle of the Percussion Wave.* When the heart's action is feeble or sudden, the volume of blood small, or the resistance in the capillaries much lessened, the up-stroke of the tracing is vertical, and the down-stroke forms an acute angle with it. The dicrotic wave is pronounced, and often descends unduly low, sometimes to the base line. These are the characteristics of low tension (see Fig. 67). When the

dicrotic wave springs from a lower level than the base line of the tracing it is *hyperdicrotic*. When the dicrotic wave is wholly effaced in the succeeding up-stroke it is *monocrotic*.

FIG. 67.



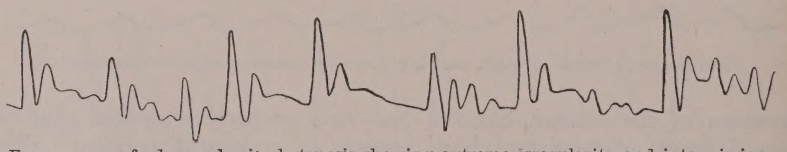
Low tension with irregularity, from cases of mitral regurgitation.

While dicrotism is commonly associated with low-tension pulses, it is occasionally met with also in high-tension pulses. Sansom says, however, that he has scarcely ever observed the conjunction of broad summit and marked dicrotism without the patient manifesting the signs of failing heart.

5. *Irregularity of the Base Line.* This occurs normally in some persons as the result of respiration, especially deep breathing. It occurs in respiratory diseases also, and in affections causing dyspnœa. Decided undulation of the base line, the curves being irregular, occurs in tubercular meningitis.

6. *Differences in the Height of Successive Percussion Waves or in their Distance from Each Other.* These are written evidences of disturbance in the rhythm of the heart. The first expresses irregularity in volume

FIG. 68.



From a case of advanced mitral stenosis showing extreme irregularity and intermission.

of successive beats, and the second irregularity in time. When this latter amounts to the omission of a beat it is called intermission. All these changes are shown in Fig. 68.

Auscultation. On examination of the arteries the stethoscope is always used. The double stethoscope is preferable, as slight pressure only must be made upon the vessels. When the single stethoscope is used some diagnostic value obtains by the character of the shock that is transmitted to the head. The arteries open to auscultation are the carotids when the neck is slightly extended; the subclavian; the innominate above the sterno-clavicular articulation; the brachial artery in the bend of the elbow, with the arm slightly extended and the crural artery just below Poupart's ligament. The normal systolic and diastolic heart sounds are heard in the carotid and subclavian arteries. The systolic sounds may be heard over the abdominal aorta, due to tension of the vessels. The diastolic sound is rarely heard in this situation. In the other vessels no sounds are heard. *Pressure*

murmur. By pressure with the stethoscope over one of the vessels its calibre is modified and a murmur created. It corresponds to the pulse, hence is systolic in time, and increases or diminishes in intensity, depending upon the amount of pressure placed upon it. Just here may be mentioned the systolic humming which is heard in children between the third month and the sixth year over the fontanelles and sometimes over the rest of the head. Osler long ago called attention to the murmur and pointed out its lack of significance in the diagnosis of hydrocephalus.

Abnormal Sounds. Abnormal sounds or murmurs are due to alterations of the blood, disease outside of the vessels causing pressure, and disease of the vessels. Murmurs from disease of the vessels, as the aorta, are discussed under the head of arterio-sclerosis or aneurism.

Murmurs may be propagated into the arteries. A systolic murmur created at the aortic orifice may be heard in the vessels of the neck and along the aorta. On the other hand, in aortic regurgitation, the diastolic normal sound in the carotid and subclavian disappears, and the diastolic murmur is not heard.

Double Sounds of the Vessels. Double sounds are sometimes heard in the crural artery under the following circumstances. (1) In aortic insufficiency; (2) in mitral stenosis; (3) in lead-poisoning; (4) in pregnancy. Vierordt is the authority who refers to these conditions having been described by Traube, Weil, and others. Duroziez' double murmur, heard when greater pressure is used by the stethoscope, occurs in aortic regurgitation when there is good compensation. Many authorities refer to this as a valuable diagnostic sign in this affection. The double sound in all instances occurs with large and quick pulse. It is probably caused by sudden collapse of the artery and the reflux blood current which is possibly an aortic regurgitation.

Murmurs due to Alterations of the Blood. They are generated in anæmia and chlorosis. They are called functional murmurs to distinguish them from murmurs due to disease of the vessels. They are systolic in time. They are soft and low in pitch, often of a musical character. The degree of loudness may vary with the position of the patient. They are increased by excitement. The loudness of the murmur increases in the course of fevers. Murmurs in the vessels apparently of functional origin are sometimes heard. The vessels are dilated without actual disease. The increased calibre favors the development of murmur by the creation of a fluid vein. Dilatation of the innominate artery sometimes takes place, giving rise to a murmur, which in loudness and character simulates the murmur of aneurism. A functional murmur is sometimes heard in the vessels, independent of disease, in cases of aortic regurgitation. The murmur is systolic in time.

Pressure Murmurs. Pressure of the stethoscope, or that caused by *disease outside of the bloodvessels.* When heard over the subclavian artery, pressure murmur may be due to adhesions or consolidation at the apex of the lung. It is more frequently heard at the left, and may be only present during full expansion of the lung. It is due to temporary pulling or bending of the artery during deep breathing. When it occurs on both sides, it is not of much significance. Murmurs in the

axillary artery, or in the arteries anywhere, when surrounded by enlarged lymphatic glands, are created by their pressure on these structures. Murmurs in the thyroid gland have been referred to (see Goitre).

Murmurs due to Disease of the Arteries. In the aorta the murmurs are due to aneurism or atheroma, or both. In the smaller vessels both conditions may be present, although atheroma is the usual one. The murmur is systolic in time, rough in character, strong, or weak. It is associated with other signs of atheroma.

Percussion. Percussion is applicable to disease of the aorta only. The methods by which it is conducted and the results of the examination will be considered in the section on Aneurism.

The Veins. The jugular veins and the cutaneous veins are alone open to examination. The femoral and the popliteal vein can sometimes be examined when the seat of disease. The ophthalmic veins are examined by appropriate instruments.

Inspection. By inspection the degree of fulness of the veins, the occurrence of pulsation, and the presence of thrombosis is ascertained. Increased fulness is due to obstruction to the flow of blood toward the heart. The increased fulness may be general or confined to the veins of one side or of one extremity. *General Increase in Size.* In the first instance there is general venous engorgement. The jugular veins, both internal and external, are seen to be distended, even in stout people. The observation can be better made by viewing the head when it is turned to the opposite side from the vein which is under examination. The external jugular can always be seen; the internal jugular when engorged. They may also be felt under these circumstances. The position of the veins can be more readily distinguished by observing their relation to the sterno-cleido-mastoid muscle. The internal jugular vein is seen in the inter-sterno-cleido-mastoid fossa, just behind the sterno-clavicular articulation. Here the jugular bulb is seen. When abnormally full it may project beyond the surface. It may be distinct in the dorsal posture. Engorgement of the external veins of the remainder of the body are not usually so readily observed, because œdema is frequently associated with it, and indeed such engorgement is generally accompanied by cyanosis, œdema, ascites, and enlargement of the liver and spleen. Because of the general fulness, there is dilatation of the right heart (which see), particularly that form which succeeds organic disease in other portions of the heart. In some instances pressure upon the cava, by an aneurism or tumor in the mediastinum, may cause increased fulness in the veins. *Local Increase in Size*, or fulness. Local increase of fulness of the veins is due to narrowing or closure of the venous trunk by pressure or by thrombosis. Here again a *mediastinal tumor* pressing upon the cava will cause abnormal fulness of the jugulars. In thrombosis of the longitudinal sinus, the veins of the skull become distended and tortuous. Enlargement of the veins of the arm or leg points to compression or thrombosis of the axillary vein or the femoral respectively. The enlargement is associated with œdema of the respective extremity. Enlargement of the superficial veins of the thorax is seen in intra-thoracic pressure from tumor or aneurism. Enlarge-

ment of the veins of both legs may be due to obstruction of the vena cava or both iliac veins. The latter is liable to occur in pelvic tumors. When there is engorgement of the portal vein the collateral circulation set up is frequently carried on through the abdominal veins. The veins are enlarged; and, in some instances, also the veins about the navel. Because of a permanent patulous condition of the umbilical vein the crown of veins—*caput Medusæ*—is formed. Enlargement of the veins of the extremities, from the causes above mentioned, must not be confounded with the unilateral or bilateral varicosity that occurs after pregnancy, after prolonged intra-abdominal pressure from other causes, or in inflammation of the veins which may have occurred in the course of septic diseases, as typhoid fever.

Pulsation of the veins. The circulation in the veins differs from that in the arteries. The blood-flow is continuous. It is modified by the respiratory movements. The modification is particularly seen in the veins of the neck. During inspiration, all of the veins empty rapidly while in forced expiration, or with strong effort, as seen in coughing, the discharge from the veins is checked and they become full and even over-distended. When the fulness of the veins is normal the respiratory alterations are not observed, except the swelling that occurs in severe coughing, as in whooping-cough. When they are abnormal as from right-sided cardiac dilatation (*q. v.*), they show a corresponding to-and-fro swelling, synchronous with respiratory movements. Upon coughing, the jugular bulb may appear as a rounded bunch between the heads of the sterno-mastoid muscle. The internal jugular may also swell and contract. Fulness of the veins is seen during the labored expiration of asthma and emphysema.

Rhythm. Alteration of the rhythm is observed in cases of pericarditis or of mediastino-pericarditis. The vessels are drawn upon and bent during the act of inspiration. They swell up at this time and empty during the expiration, directly opposite to the normal state.

The Venous Pulse. The cardiac movements also modify the movements of the blood in the veins. They cause rhythmical pulsation, or the venous pulse. This may be communicated from the carotids underneath or occur in the vessels. The so-called true and false pulses are thus produced. The true venous pulse is divided into the *negative* and *positive* pulse, the former being the pulse of health, the latter the pathological venous pulse. The negative venous pulse is presystolic and can only be seen in the external jugulars. The vein collapses during the systole and distends before the systole, hence is presystolic. This may be observed by inspection, keeping in view also at the same time the apex or the carotid pulse. The systolic collapse occurs quickly. The presystolic follows slowly, with an appreciable interval between the two. The presystolic distention occurs during the time that the auricle is filled with blood; the collapse occurs when the auricle is empty, that is, during the ventricular systole. When the auricle is distended, the flow of blood from the veins is impeded and hence the jugulars are overfilled. When the auricle is empty the flow of blood from the veins is favored, hence the vein collapses (the systole). Sometimes it is extremely difficult to recognize the normal or negative venous pulse

on account of undulations in the veins, produced by the blood-flow and transmitted carotid impulse.

The positive venous pulse is systolic in time. It is pathognomonic of tricuspid regurgitation (*q. v.*). When the right ventricle contracts the regurgitant blood-wave is transmitted into the cava through the incompetent valves. It appears in the internal jugulars or their bulbs, because of the direct course of the innominate and right jugular from the cava. Subsequently the left may become affected. If the valve in the vein is competent a systolic regurgitant wave ensues there. The pulsation of the enlarged bulb is seen in the inter-sterno-cleido-mastoid fossa. Usually the valve is insufficient, or rapidly becomes so, and the systolic back-wave therefore extends upward. The same wave is transmitted to the veins of the liver, causing systolic swelling and diastolic collapse of the liver. These conditions are produced, as previously mentioned, in right-sided dilatation of the heart, providing there is moderate force and slowness of the heart's action. When the heart becomes very weak and rapid the pulsations disappear.

The negative, true, or normal pulse is distinguished from the pathological or positive pulse, and from the transmitted pulsation, by its time. Comparison of the apex beat, or the carotid pulse of the opposite side, shows the collapse to occur during the systole in health, whereas in the other conditions fulness takes place during the systole. The patient should hold the breath, as increased respiratory movement will modify the venous pulsation. The imparted or false pulse, transmitted from the carotids, can be distinguished by stopping the flow of blood by means of pressure of the finger in the middle of the neck upon the vein after it has been emptied by pressure upwards. If the pulsation is communicated, the vein remains empty in the portion nearest the heart, and fills up in the peripheral portion. In the positive pulse the portion near the heart is filled. Diastolic collapse has been spoken of under the head of Pericarditis. In congenital heart disease the systolic venous pulse may sometimes be seen, but is extremely rare.

In the other affections Quincke has described venous pulse in the hand and back of the foot with the capillary pulse in aortic regurgitation and in anæmia. It is probably only the arterial pulse propagated through the capillaries. The systolic true pulse previously described may be seen in the veins of the face, in the cutaneous veins of the arm, in the internal mammary veins, and in the inferior vena cava.

Thrombosis of the Veins. This is usually detected by palpation, and occurs most frequently in the femoral vein. The vein is transformed into a firm, round cord, and is distinguished from the artery by the absence of pulsation. Thrombosis in these veins and in the iliac veins higher up occurs in acute infectious diseases and in the debility of the aged. Dropsy in the area of distribution of the veins is perceived.

Auscultation. In health no sounds are heard. Two conditions contribute to the creation of a murmur in the veins: 1, change in the character of the blood; 2, dilatation with the occurrence of positive venous pulse.

The Venous Hum. In anæmia and chlorosis, sometimes in healthy patients a hum or murmur or buzzing sound is heard over the jugular

veins. It is louder on the right side than on the left. It is soft and low in pitch, and may be musical; it has been described as humming or whizzing. It is continuous. For its detection a double stethoscope should be used, as pressure increases it, and the patient should not turn the head aside. It is increased when this position is taken. The murmur is modified by the respiration and by the cardiac action. It is louder in deep inspiration when the blood is going more rapidly to the thorax. It is also louder in the upright position. It is frequently louder during the diastole. The increased loudness at these periods occurs because, from the suction action during inspiration and during the diastole, the blood is more rapidly drawn toward the heart. The murmur is caused by the flow of blood from the narrow jugular into its wider bulb, on account of which a fluid vein is produced. Similar murmurs are heard in other veins, as in those of the extremities when the anæmia is profound. They are then stronger during the diastole of the heart.

The Data Obtained by Inquiry.

The Subjective Symptoms of Heart Disease.

A. Symptoms Referred to the Heart. 1. *Pain.* While pain in the region of the heart may be a symptom of disease of that organ or of the pericardium, in the large majority of instances it is due to other causes. The physician is frequently consulted by the anxious patient on account of pain, other than heart pain, referred to this region, or more precisely to the fifth or sixth interspaces on the left side. The causes of such pain are various: 1, neuralgia; 2, pleurodynia; 3, myalgia; 4, local pleurisy; 5, periostitis. The *neuralgias* may be associated with points of tenderness, which are usually the seat of the greatest intensity of the pain. These points of tenderness correspond with the positions at which the nerves have their exit through the fascia to the surface, and are found along the sternum, in the course of the mid-axilla, and along the vertebræ. The pain is paroxysmal, occurs at variable periods of the day, and in anæmic subjects or in the course of neurasthenia. It may precede the development of herpes zoster. In these cases the exact nature of the pain is not known until the eruption appears. In gout or diabetes we may have local neuritis, which causes neuralgic pain in this situation.

Pleurodynia, which is thought to be an affection of the pleural nerves, is more general. The pain is increased by pressure of the finger-tips, although it is not localized. It is relieved by pressure of the whole hand. In *myalgia*, which is seen so frequently in phthisis on account of severe coughing, in rheumatism and in debilitated subjects generally, the pain is more or less diffuse, interferes more or less with movements of the chest, is relieved by uniform general pressure, and is usually associated with myalgia in other regions. The pain of *pleurisy* is recognized because it usually inhibits the act of breathing, is associated with cough, and friction sounds may be detected. *Periostitis.* In disease of the ribs of the præcordia the pain is associated with tenderness and swelling. One or more of the costo-sternal articulations may be

extremely tender. The pain and tenderness are due to the periostitis of syphilis or to that which follows typhoid fever. In one of my cases the rib had to be resected. It may be due to the internal pressure and erosion of ribs in aneurism. The same affection may cause neuralgic pains in the nerves. *Abscess.* Pain in this region in rare instances may be due to localized tuberculous abscess between the pericardium and the walls of the thorax. One such case was under my care. The abscess developed secondarily to empyema and occupied the præcordial region, causing bulging. The pain was intense, and was only relieved after the caseating pus was removed by incision.

Pain in the *epigastrium* is often held to be due to cardiac disease. It is usually due to gastralgia, or, as it is sometimes termed, cardialgia. It is recognized by the location of the pain and its association with gastric symptoms, as flatulency, weight, fulness, and acidity. In gastric ulcer the epigastric pain is localized, accompanied with tenderness on pressure, and increased by food.

Pain in Disease of the Pericardium. Pain in the region of the heart is sometimes due to affections of the pericardium. *Pericarditis* is the most common. While centralized in the heart region, it may radiate to the left shoulder and extend down the arm. It is paroxysmal and may have some of the characters of angina. It is increased by movement, by pressure, and by the action of the diaphragm. The patient is often obliged to sit up in bed and suffers from orthopnœa. A pericardial friction sound is usually detected. *Pain due to disease of the aorta. Acute inflammation of the aorta* is also the cause of cardiac pain. The pain extends along the course of the aorta, may be referred to the sternum, and extends along the spine. The pain is severe, causing an anxious countenance and an expression of extreme suffering. In gouty subjects with *atheroma,* pain may occur in this situation in paroxysms. There is usually valvular disease at the aortic orifice. Similar pain occurs in syphilis and in alcoholic subjects, and may be due to malaria. It is a visceral neurosis, or a form of neuralgia.

Pain in the region of the heart is frequently due to *aneurism.* The pain is usually due to pressure of the aneurism upon adjacent structures. If the bone is pressed upon and erosion is going on, the pain is of a boring character, localized at one point. It has been previously referred to. In aneurism alone, without pressure, the pain is of a dull aching character, increased by movement, relieved by rest. When nerves are pressed upon pain may be acute and of the nature of a neuralgia. It may follow the course of the nerves and be associated with numbness or sensations of tingling. The long duration of the pain, its localization, and its aching character are sufficient to exclude angina pectoris. When the pain is unilateral it may be due to pressure of an aneurism upon the nerves at their exit from the canal; the pain extends along the course of the intercostal nerves. It is severe and burning, but there are no localized points of greater intensity. The pain may extend down the arms, and when the abdominal aorta is affected it may extend down the legs. In the course of rupture of the aneurism the pain is sudden and sharp. Death, however, ensues quickly, so that the pain will rarely be complained of.

Pain in Disease of the Heart. Three forms are seen: 1, pain due to disturbances of the rhythm; 2, pain due to valvular disease; 3, pain due to angina pectoris.

Disturbance of the Rhythm. Palpitation, intermission, and irregularity of the heart occur in the large majority of cases without pain. Paroxysms of palpitation are sometimes attended with severe præcordial pain and distress. This occurs in the reflex palpitation, which, as will be seen, is due to disease in other situations; in the palpitation of Graves' disease and of anæmia. The palpitation of organic disease is induced by exertion. The rapid action of the heart is painful and the throbbing is complained of, causing distress.

While intermission and irregularity may continue without pain at times, the patient is conscious of this disturbance of the rhythm and complains of the stoppage, which then is attended by distress, sometimes amounting to severe pain. This is particularly the case when the heart action is tumultuous, as the disturbance of rhythm seen in pericarditis and in valvular disease.

Pain due to Valvular Disease. In disease of the aortic valves pain is of more frequent occurrence than in other valvular lesions. It is usually complained of in the region of the aorta at the base of the heart, and is aggravated by exertion. (See Atheroma.)

Pain due to Angina Pectoris. Heberden was the first to describe the attacks of angina pectoris, which, in its typical form and in association with disease of the heart, is not of common occurrence. The pain of angina is severe and is associated with the most intense anguish. It comes on suddenly, and may occur in paroxysms. The patient realizes that the pain is in the heart, and complains of feeling as if the organ were held in a vise. From the heart it radiates to the neck and down the arms. It particularly extends to the left arm, and may be severe in the wrist or in the ends of the fingers. With the pain there is a sense of impending death, with sinking and depression. The pain lasts but a few seconds or minutes, and during that time the face of the patient becomes pale or of an ashen hue, perspiration breaks out on the forehead, the extremities become cold, the breathing is short. Prostration usually follows the attack, but the præcordial distress disappears entirely. The attack may occur in patients who are free entirely from organic disease of the heart. It is most commonly, however, associated with some lesion. The lesions frequently found are disease of the coronary arteries, atheroma of the aorta, aortic valve disease, and myocarditis with fatty degeneration. It occurs after middle life, and is more frequent in males. It may occur without exciting cause, or follow undue exertion, exposure to cold, mental excitement, or profound emotion. The points upon which the recognition of the nature of the attack can be made are: (1.) The seat of the pain. This is usually behind the middle of the sternum, or the lower part, and more to the left than to the right. From thence it extends to the posterior portion of the axilla or it may radiate up to the neck. In some instances it extends to the occiput. Frequently the pain extends to the left arm as far as the elbow or even to the fingers. It may extend to the abdomen or to the right arm. I have seen it affect both arms. It is not influenced by external pressure. (2.) The sense of con-

striction with the indescribable torture of an intense pain are most characteristic. (3.) The respirations are shallow, or even may cease, but there is no dyspnœa. (4.) The patient is terrified and restless. (5.) The pale face, extremely anxious countenance, the cold sweat on the forehead, make a striking picture, which when once seen can never be forgotten. (6.) Such extreme depression and sensation of impending death occur in no other affection. Particularly characteristic is the immediate relief, without hysterical manifestations or dyspeptic symptoms of any kind, which follows an attack. (7.) During the attack the frequency of the pulse is not much influenced, and the action of the heart may be uniform and regular. The tension of the pulse is increased during the attack.

Some authors refer to various grades of angina, and call all forms of præcordial pain and oppression, with radiation of the pains to the arms and neck, mild forms of angina. Such attacks have often obvious exciting causes in disturbance of digestion and in emotional excitement. When associated with increased arterial tension and signs of arterio-sclerosis they may be of an anginoid nature. The greatest difficulty exists in distinguishing them from pseudo-angina. Hysterical or pseudo-angina can be distinguished only with extreme difficulty. It is likely to occur much more frequently than true angina. One attack seems to predispose to others. It occurs in females who present other symptoms of hysteria. It occurs usually before forty years of age. The attacks are most frequent at night, and may be periodical. They are particularly associated with menstrual disorders. The pain is less severe and the oppression is not so marked in pseudo-angina; coldness of the hands and feet, with the occurrence of syncope, or a general feeling of sinking, are common symptoms. The pain is of long duration and is associated with great agitation. It is preceded by neuralgia, and neuralgic pains persist after the attack. There is low tension, feeble second sound and soft arteries. The disease is never fatal. In one of my patients, attacks of hysterical hæmoptysis alternated with the anginal attacks.

2. *Palpitation.* In palpitation the patient is conscious of the action of the heart. Although it may occur in organic disease, it is more frequently due to diseases outside of the heart.

Symptoms. The symptoms vary in degree. In mild forms the patients may complain of a fluttering or a sensation of sinking in the præcordial region. In the more severe forms the heart beats violently against the chest. The arteries throb, the action of the heart is increased, and the area of impulse against the chest wall is enlarged and visible. The patient complains of distress in the præcordial region. The pulse may be increased to 150. In nervous palpitation, the face becomes flushed, and after the attacks large quantities of urine are passed. Sometimes, in this form of palpitation, exertion relieves the attack. On examination, the sounds are found to be normal, but they are clear and metallic in character. The diastolic sounds are greatly accentuated. If anæmia is present, murmurs due to that condition are increased in intensity. The attack may last but a few minutes or continue for hours.

(*a*) It is most common in cases in which the nervous system generally is in a state of increased excitability. Attacks of it occur at puberty

and at the menopause. It is very common in hysteria and neurasthenia. It follows emotional disturbance. It is more frequent in women.

(*b*) It is due to the action of toxic substances, as tobacco, tea and coffee, and alcohol.

(*c*) From strain and over-exertion, particularly if associated with excitement, palpitation may occur and continue for a long period. This is the form of irritable heart, described by Da Costa, common in young soldiers during the war.

(*d*) In valvular disease of the heart when compensation fails, and in myocarditis, attacks of palpitation occur. They usually then distinctly follow exertion.

3. *Intermission and Irregularity.* When the patient feels the alteration in rhythm, it is usually due to nervous disturbance. In organic disease usually it is not experienced by the patient. Although not a subjective symptom alone, it may be well to speak of irregularity in this connection.

Arrhythmia is the general term applied to irregularity of the action of the heart. When the heart intermits, that is when one or two beats are dropped at intervals of half a minute, a minute, or longer; when the beats are unequal in volume and force, or occur at unequal distances in time, the heart's action is irregular. The causes of disturbance of the rhythm have been classified by Baumgarten[1] as follows: 1. Central causes in the medulla either from organic disease, as hemorrhage or concussion, or from psychical influences. 2. Reflex influences, as in dyspepsia and diseases of the liver, lungs, and kidneys. 3. Toxic influences—tobacco, coffee, and tea are common causes; various drugs, such as digitalis, belladonna, and aconite. 4. Changes in the heart itself. Mural changes, as dilatation, fatty degeneration, and myocarditis; changes in the cardiac ganglia; sclerosis of the coronary arteries.

It must not be forgotten that both irregularity and intermittency may occur in persons otherwise in good health and continue for a long period of time without any evidence of arterial or cardiac disease. (For the varieties of arrhythmia see The Pulse.)

B. Symptoms referred to the Circulation. 1. *Pulsation of the Arteries.* Pulsation of the arteries, especially the carotids, the abdominal aorta, and the brachial arteries occurs in anæmia and is common in emotional disturbances. Such pulsation, as of the abdominal aorta, may be reflex from organic disease in the vicinity. Similar localized pulsation in the innominate arteries may be mistaken for aneurism. The pulsation that attends organic heart disease may be due to hypertrophy of the heart, but is particularly characteristic of aortic regurgitation.

2. *Hemorrhages.* In the description of valvular lesions it will be seen that hemorrhage from the lungs occurs quite frequently in disease of the mitral valve. The hemorrhage may be due to congestion, to actual rupture of the vessels, or to hemorrhagic infarct (see Pulmonary Hemorrhage). It may simulate hemorrhage due to tuberculosis.

[1] See Transactions of the Association of American Physicians, vol. iii.

3. *Cyanosis.* Cyanosis is a symptom of common occurrence in the course of organic heart disease (see page 72).

4. *Dropsy* (see page 92). The dropsy of heart disease occurs after failure in compensation in the course of valvular disease and in dilatation of the heart. It may disappear entirely, if the conditions are improved, or become permanent and progressive. In general, it may be said to be distinctly a phenomenon of mitral regurgitation and secondary tricuspid regurgitation. It occurs in less degree in mitral obstruction, and still less in disease at the aortic orifice.

C. Symptoms referred to the Lungs. The chief subjective symptom is dyspnœa. Dyspnœa, due to disease of the heart, is clinically divided into (1) dyspnœa that is set up or increased by exertion; (2) paroxysmal dyspnœa; (3) orthopnœa; (4) rhythmical dyspnœa, or Cheyne-Stokes respiration. The dyspnœa of effort takes place after the slightest exertion. In paroxysmal dyspnœa the attack comes on without apparent cause. It must be distinguished from the paroxysmal dyspnœa of asthma or emphysema. The physical signs of lung disease usually point to the latter. The paroxysmal dyspnœa of heart disease is attended by more violent efforts of breathing than the physical state of the lungs warrants, and the difficulty attends both inspiration and expiration. Wheezing is not so marked as in forms of asthma. There is some obstruction to the outgoing of air, but, on account of air-hunger, all the efforts of the patient are used to fill the chest. In paroxysmal dyspnœa, if the patient is placed in a comfortable position, the breathing generally is quieted, provided there is no lung or pleural complication. The position does not modify the severe dyspnœa of asthma or emphysema. Orthopnœa has been described previously (see page 282). (For Cheyne-Stokes respiration see page 239).

Cough. Cough is of frequent occurrence in heart disease. The causes are various. It may be due to pressure upon the bronchus or the pneumogastric nerves, as in pericardial effusion. It may be due to the congestion of the lungs which occurs in failing compensation. A low-grade bronchitis may develop on account of passive congestion, causing cough. If hemorrhagic infarcts take place, cough may be present. It attends the broncho-pneumonia that follows. In the cough that occurs from pressure of an aneurism, a metallic brassy cough is created, which occurs in paroxysms and may be associated with alterations in the voice. The clanging cough may result in the expectoration of blood-tinged sputa, which is frequently due to the gradual rupture of the aneurism.

D. Symptoms referred to the Brain. The symptoms are usually due to disturbance of the cerebral circulation, either because insufficient blood is supplied to the brain or because improperly oxygenated blood is supplied. Vertigo, faintness, and languor are complained of in the first instance. Dulness, stupor, and moderate delirium may occur in the later stages in the second instance. In the course of organic heart disease, *epilepsy*, or *epileptiform convulsions* may arise on account of embolism or thrombosis. *Chorea* is of common occurrence, but bears causal relation to the organic disease. *Coma* in the course of heart disease may be due to hemorrhage into the brain, embolism, or to thrombosis. Hemorrhage occurs in patients in whom, at the same time,

there is usually found hypertrophy of the left ventricle, atheroma of the artery and renal disease. Embolism occurs in valvular disease, particularly in aortic regurgitation and mitral obstruction. With or without coma we may have the occurrence of paralysis for the same reason.

Thrombosis in the course of heart disease is usually due to disease of the bloodvessels rather than disease of the heart itself, although weakening of the heart, as in dilatation, is a predisposing factor to the development of thrombosis.

E. Symptoms referred to the Alimentary Canal. In the course of organic heart disease, dyspepsia and forms of gastritis are of common occurrence. Patients complain of indigestion of various forms, or of nausea and vomiting. While water-brash and flatulence are caused primarily by the condition of the heart, they may in their turn more frequently cause symptoms of palpitation and cardiac distress. These gastric difficulties are more particularly seen in diseases of the auriculo-ventricular valves and are associated with congestion of other abdominal viscera.

F. Symptoms referred to the Throat. The patient may complain of pain in the throat. This may be paroxysmal, and is sometimes said to be due to angina pectoris. Hoarseness or modifications of the voice are occasional symptoms of pericarditis. They are of frequent occurrence in the course of aneurism due to pressure upon the recurrent laryngeal nerves.

G. Symptoms referred to the Kidneys. The kidneys are intimately related with the heart at a distant point in the circulation, and are the frequent seat of changes due primarily to disease of the central organ of circulation. The changes in the urine will be referred to again; suffice it to say that in the course of mitral and tricupsid disease and dilatation, scanty urine, of high color, loaded with urates, containing a small amount of albumin, is quite common and indicative of *passive congestion* of the kidney. It may result in cyanotic induration or interstitial nephritis. On the other hand, the urine may be of low specific gravity and pale in color. There may or may not be traces of albumin. The change is due to a granular, *contracted kidney*, which is associated with hypertrophy of the left ventricle and arterial sclerosis. Bloody urine is usually due to *renal embolism* when it occurs suddenly in the course of organic heart disease. It may be due to the emboli that are found in septic endocarditis. Renal disease in all forms may complicate disease of the heart. (See Kidney Disease.)

The Subjective Symptoms of Arterial Disease.

The patient may complain of an increased amount of blood in a part, or of a lessened amount. Thus the symptoms of anæmia in a part, as vertigo and giddiness, or of flashes of light, may attract attention. (See Cerebral Thrombosis.) All the symptoms of deficient supply of blood to the brain may be present. The feet are cold for the same reasons. The diseased vessels cause blood to be kept away from the area. Pain is common only when atheroma or aneurism is present (*q. v.*). Throbbing or pulsation is complained of. It may be a striking hysterical or

neurasthenic feature. The abdominal aorta is frequently thus affected. The pulsation may be constant or intermittent. There may be dyspeptic symptoms. The pulsation of the carotids may cause abnormal sensations in the head, and the beating be a source of extreme annoyance.

Pericarditis.

Inflammation of the Pericardium. The inflammation may be acute or chronic. It is also divided according to the nature of the inflammation into simple fibrinous inflammation and inflammation with effusion. The effusion may be serous, bloody, or purulent, depending upon the nature of the inflammation. Pericarditis, either acute or chronic, is also divided into primary or secondary pericarditis. The primary form is of extremely rare occurrence. Indeed, it may well be doubted whether, in common with the inflammations of serous membranes in general, pericarditis is ever primary, or so-called idiopathic, in origin.

1. *Pericarditis* may be of *local* origin by extension from processes which have originated in organs in the vicinity of the pericardium. It may follow a pleurisy and partake of the nature of the primary pleural inflammation. It often attends empyema, either from extension of the infection to the pericardium, or from rupture into the pericardial sac. It follows all forms of inflammation of the mediastinum. Disease of the ribs adjacent to the pericardium may set up pericarditis. Inflammations below the diaphragm frequently give rise to pericarditis. Peritonitis, when general or local; sub-diaphragmatic abscess; suppurative gastritis, with perforation of the stomach; abscess of the liver; suppurating hydatid, and other forms of suppuration below the diaphragm, also lead to pericarditis.

2. *In General Diseases.* The general diseases which are the cause of inflammation of the pericardium are those which usually give rise to inflammation of serous membranes. They are: Infectious diseases, particularly scarlet fever, measles, erysipelas, and typhoid fever. All forms of septicæmia may be attended by inflammation of the pericardium. Tuberculosis is a frequent cause of pericarditis. Inflammation of this membrane frequently arises in the course of rheumatism. It may occur in the course of the disease, or attend some of the affections which are associated with, if not themselves of a rheumatic origin, such as acute tonsillitis. In the course of certain dyscrasiæ the pericardium is frequently the seat of inflammation. This is particularly the case with scurvy. It occurs also frequently in Bright's disease and may be the first manifestation to the patient of this disease. This is particularly the case in the chronic form of Bright's disease. It occurs in the course of gout. *Age.* The various forms of pericarditis may occur at any age, although that which attends scarlatina and rheumatism occurs in early life, while late in life it is an attendant upon chronic Bright's disease and gout.

While rarely an attendant upon diseases of the heart, except as a coincidence, it is said to occur after ulcerative endocarditis, after myocarditis, and during the course of aneurism of the aorta.

Acute Fibrinous, or Plastic, Pericarditis. This is probably the most common form that is seen. It is particularly the variety that occurs in the course of Bright's disease and rheumatism. It may be wanting entirely in symptoms. An examination of the heart in the routine of duty may reveal its presence by physical signs. In the course of either of the above-mentioned diseases in which it occurs secondarily it may happen that the temperature rises a little higher than it should, or that convalescence does not take place so rapidly as we should expect from the amelioration of other symptoms, such as the joint inflammations in rheumatism. On examination of the pericardium the friction sound is detected. In other instances the patient may complain of pain in the region of the heart. It is usually localized in the fourth or fifth interspace. It is not very severe and not influenced by pressure. Sometimes the pain is complained of at the xiphoid cartilage. In rare instances it may resemble angina. The pain and the occurrence of fever further call attention to the heart.

Physical Signs. Inspection. Nothing unusual is observed, although the heart may be seen to beat more violently against the chest wall. The impulse is diffused. By *palpation* a friction fremitus may be detected, due to the rubbing together of the roughened pericardial surfaces. It is not always present. It may be felt when the whole hand is laid over the præcordia, or when palpation by the tips of the fingers alone is resorted to. It is most marked over the right ventricle, particularly in the fourth interspace, and is increased when the patient leans forward.

Auscultation. The friction sound is usually present. It may be present while the fremitus is absent; but on the other hand, if the fremitus is present we can always hear the friction. It is heard over the region where the fremitus is felt. *Character.* It is a to-and-fro rubbing or grating sound; sometimes it is quite high in pitch; it may be of a creaking character. It gives one the sensation of being near to the ear. It may be modified by the pressure of the stethoscope and by the position of the patient. It may be heard in the erect and disappear in the recumbent posture. *Position.* It is localized, and not transmitted away from the heart. It may be heard along the course of the sternum. It is usually heard in the third or fourth interspace, but may be heard as high as the second, adjacent to the sternum in either interspace. Sometimes it is heard at the second costal cartilage on the right, rarely at the apex. The point of maximum intensity varies with the position of the patient. *Time.* It is both systolic and diastolic. In some cases it may be only systolic in time, or it may be of a galloping nature, representing three sounds during the cardiac cycle.

Diagnosis. Acute pericarditis without effusion is not recognized generally because it has not been sought for. In the larger number of cases, as previously intimated, there have been no indications of disease of the pericardium during life. If sought for, however, the diagnosis is usually easy. The *pericardial friction* may be mistaken for an *organic heart murmur* or for *pleural* or *pleuro-pericardial friction.* It is often difficult to distinguish the to-and-fro friction from the murmurs of double aortic disease. If attention is paid to the general and local

phenomena the mistake is not likely to be made. The location of the murmurs in organic heart disease, the direction of the transmission, the character of the murmur, the peculiar character of the pulse and the secondary effects upon the muscles of the heart, point to the diagnosis of a valvular lesion. The pleuro-pericardial friction which simulates pericardial friction usually occurs in the course of phthisis or pleuro-pneumonia. It is modified by respiratory movement: (1) It may disappear or lessen notably if the breath is held; (2) a full expiration may cause its disappearance. While it is of cardiac rhythm it is modified by the respiratory rhythm, so that on inspiration it is usually more marked. The pleuro-pericardial friction is not strikingly modified by position. *Pleural Friction.* This is of respiratory rhythm and ceases with cessation of breathing. The pericardial friction persists if the breath is held.

Pericarditis with Effusion. I know of no affection which is more frequently overlooked during life than pericardial effusion. This occurs because its development takes place without symptoms. In plastic pericarditis we have referred to the occurrence of pain. This may occur before the effusion in the latter form, but is usually moderate. As with dry pericarditis, however, it may, in rare instances, be very severe, anginous in character, and be increased by pressure over the heart or on the pit of the stomach.

The *symptoms* are usually due to the presence of fluid in the pericardium. On account of it pressure symptoms arise, and the well-known physical signs take place.

1. General Symptoms. Rarely, however, a few general symptoms occur. These are usually cerebral. Delirium, which may be moderate or maniacal, has been reported in a number of cases. It must not be confounded with the delirium which occurs in the course of acute rheumatism with hyperpyrexia. In addition, choreiform movements have been described. They may, however, be of rheumatic origin. Other cerebral symptoms, as hemiplegia and convulsive attacks in the course of pericarditis, are probably due to an associated endocarditis, causing embolism, the endocarditis not having been recognized. In some cases albuminuria is found.

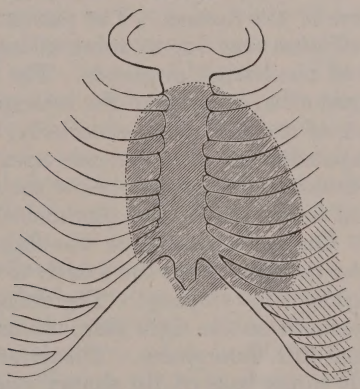
2. The Pressure Symptoms. *Dyspnœa* is the most common. The degree depends upon the amount of effusion. If the latter is large there may be extreme orthopnœa; if the effusion is present for a considerable time, it may give rise to no symptoms. *Dysphagia.* In large effusions this may occur on account of pressure upon the œsophagus. *Altered Cardiac Rhythm.* The effect of the effusion upon the heart is to interfere with its action, which, although usually regular, becomes on the slightest exertion or the least excitement, irregular or subject to severe attacks of palpitation. The heart's action is increased in frequency; when the effusion is very large it may be not only irregular, but also intermittent. *Aphonia* may occur from pressure upon the recurrent laryngeal nerve. *Cough* of an irritative character is sometimes noted, from pressure upon the veins within the thorax. The pulsus paradoxus may be present.

3. Physical Signs. *Inspection.* There is bulging of the præcordia, particularly in children. The ribs and interspaces are prominent. In adults the interspaces are even with or distended beyond the surface of the ribs. The enlargement may extend to the antero-lateral region of the left chest. The large effusion interferes with expansion of the lung on the left side, and hence movement is diminished. In such cases the epigastrium may be prominent on account of dislocation downward of the diaphragm and liver. The apex beat is absent or faintly seen dislocated upward and to the left. It may be seen in the fourth interspace, or a faint impulse observed in the second and third interspaces beyond the mid-clavicular line.

Palpation. The impulse is feeble and diminishes progressively as the effusion increases. The position of the apex determined by inspection is confirmed. The pericardial friction which may have been present at first disappears with the effusion. Fluctuation may be detected in large effusions.

Percussion. The area of pericardial dulness is increased. The increase of area usually is found in all directions, although increase of the

Fig. 69.



Percussion dulness in pericardial effusion; the lower and left margins left undefined, owing to their having been inseparable from the dull percussion of the abdomen and of the left pleura. (Gairdner.)

dulness upward and to the left is very common. It may extend as high as the second rib. As pointed out by Rotch, dulness in the fifth right interspace is common in effusion. The triangle formed by the right border of the heart and the right lobe of the liver is dull instead of resonant. The dulness in large effusion includes the axillary region, so that it may simulate a pleural effusion. The dulness, however, does not extend below the eighth rib in this region, whereas, in pleural effusion, dulness always extends to the bottom of the pleural sac. In pericardial effusion, when it is large, the semilunar space, or Traube's line, is obliterated.

Auscultation. On auscultation the sounds are feeble and distant. They may be scarcely heard at all over the præcordial region. The sounds at the base of the heart are diminished in intensity. If a fric-

tion sound was heard at the beginning it disappears entirely as the effusion is poured out. In moderate effusions the friction may be heard when the erect posture is assumed. The change in the rhythm of the heart which attends pericarditis is noted.

It must not be forgotten that the physical signs, and especially the change in impulse and the area of præcordial dulness, are modified by the position of the effusion. Accumulations occur behind the heart or above it, and in these situations interfere least with the displacement or the enfeeblement of the apex beat. The area of dulness, however, is increased upward. In cases of large effusion the compression of the lung may cause bronchial breathing to be heard posteriorly or in the axillary region. In a case under my care the diagnosis of pericardial effusion was readily made, but the enormous effusion so markedly simulated an effusion into the pleural cavity that both serous cavities were believed to contain fluid. Aspiration was performed in the sixth interspace in the anterior axillary line. The fluid was removed from the pericardium, as was afterward determined. During life the pressure signs of laryngeal stridor, difficulty of deglutition, and extreme dyspnœa, were present. Early vomiting, epigastric pain and tenderness, slight delirium, albuminuria, and an excessively weak, rapid pulse occurred in the course of the disease. The patient was a male, twenty years of age. The effusion was due to tuberculous pericarditis, secondary to tuberculosis of the bronchial glands. The physical signs were: prominence of the præcordia, bulging of the interspaces on the left side, diminished expansion of the left side—anteriorly, laterally, and posteriorly; increased expansion at the extreme apex of the lung. On palpation the vocal fremitus was absent below the second interspace in front, below the third in the axilla, and diminished below the spine of the scapula behind. On percussion there was dulness from the second left rib in front to the margin; from the fourth to the eighth rib in the axilla; below the eighth rib, tympany. The dulness extended beyond the margin of the sternum on the right side, almost to the right nipple line, in the fourth and fifth interspaces. Posteriorly, dulness from the middle of the scapula to the base of the thorax, except along the vertebræ, where, from the seventh to the ninth rib, there was tympany. The physical signs of pericardial effusion on auscultation were marked. In the axilla there was absent breathing. There were bronchial breathing and bronchophony behind from the spine of the scapula to the base along the vertebræ. They were most marked opposite the angle of the scapula where the above-noted tympany was recorded. In the mid-scapular line the breathing lessened from above downward, and was absent at the base. It is seen that the physical signs of pleural effusion were present posteriorly and laterally, due to the enormous effusion. At the autopsy the pericardium was found to contain sixty-four ounces of fluid. Pleural effusion may be excluded in similar cases by the absence of dulness in the axillary region below the eighth rib; by increase in dulness beyond the right edge of the sternum; and at the same time by the absence of signs indicating upward dislocation of the heart.

The general phenomena that attend pericardial effusion depend upon the nature of the primary disease and the character of the fluid. In

tuberculous pericarditis, emaciation, irregular fever, sweats and prostration ensue. In *purulent pericarditis* there may be recurring chills with a temperature range decidedly intermitting, along with other phenomena of purulent accumulation. In a case recently seen the patient was extremely debilitated and prostrated on account of pneumonia following influenza. He was extremely anæmic, and the blood-count showed diminution of red cells one-half without other particular change. Every fourth day after a chill the temperature would rise to 103° or 104°. A friction sound was detected after the second chill. It disappeared, but the physical signs of effusion could not well be made out. From the first the heart's action was so weak that the sounds were scarcely discernible. At the autopsy four or five ounces of pus were found in the pericardial sac, the purulent accumulation in this situation being the only lesion to account for the symptoms.

Diagnosis. Pericardial effusion must be distinguished from dilatation of the heart. This is not generally difficult, if the patient has been under observation during the development of the disease. The impulse is not always absent in dilatation; although feeble and diffuse, the expansile shock of the impulse is more distinct than in dilatation. Fluctuation may be detected. The area of dulness in dilatation does not extend upward except in cases in which the right auricle is enlarged. The dulness does not extend downward in dilatation without similar dislocation of apex beat or of impulse. The shape of the dulness differs. In dilatation the dulness is square in shape; in effusion it is triangular or pear-shaped, with the base downward. In dilatation the sounds are accentuated, and are of a valvular character; in effusion they are muffled. Dilatation does not cause the pressure symptoms that occur in effusion. In pericardial effusion Bamberger's sign is of importance. When the patient is sitting upright an area of dulness about the size of a silver dollar can be marked out at the angle of the scapula. Over it, dulness, increased fremitus, and bronchial breathing are made out. If the patient leans forward the dulness disappears with the other signs of consolidation, to return when he sits upright.

In pericarditis with effusion, after its absorption the friction sound may return again. It is of diagnostic significance to have change of rhythm and character of the sound from day to day, or of its degree of loudness on movement of the patient. Often it may disappear entirely and all signs of pericardial inflammation subside. In plastic pericarditis and pericarditis with effusion, adhesions to the pericardium may take place.

Effusions into the pericardial sac of serum, of blood, or of air, may take place without previous inflammation.

Hydro-pericardium. This may occur in the course of general dropsy from kidney or heart disease. It may not prove fatal of itself, but when associated with effusions in the pleural sac, contributes to the orthopnœa, on account of which death takes place. Rarely after scarlet fever, effusion into the pericardial sac may be the only dropsical symptom. The physical signs are those of effusion. It is not attended by fever. It is frequently overlooked, because investigation beyond the pleura is not made after an effusion into that cavity has been found.

Hæmo-pericardium. This occurs on account of rupture of an aneurism of the first part of the aorta, of the heart itself, or of the coronary arteries. Wounds of the pericardium and heart cause hæmo-pericardium. The extension of the ulceration of malignant endocarditis to the surface may cause gradual effusion of blood. The physical signs are those of effusion. Death usually takes place before there has been time for an examination of the patient sufficient to determine its presence. Rapid heart failure due to compression is the cause of death. In the case previously referred to above, and in cases of rupture of the heart, the patient may live for many hours with dyspnœa and progressive weakening of the heart. In tuberculosis and cancer the effusion is frequently blood-stained.

Pneumo-pericardium. This occurs very rarely, and is due to perforation from without by stab-wound, or perforation from the lung, œsophagus, or stomach. A purulent exudation may undergo decomposition, causing an accumulation of gas. If it arises from perforation acute pericarditis is set up. The accumulation of gas causes tympany over the movable area of percussion dulness. The most striking sign is noted on auscultation. Churning, splashing, or metallic sounds are heard, drowning the feeble heart-sounds. Death usually occurs quickly.

Adherent Pericardium. Chronic adhesive pericarditis usually follows the acute form. The *physical signs* are, on inspection, indrawing of the interspaces with systolic contraction of the ventricles; even the ribs are said to be drawn in. This indrawing is most marked at the apex, and must not be confounded with the retraction that occurs in the third and fourth interspaces with the ventricular systole. At the same time that the retreat of the surface takes place, if the hand is placed on the heart a systolic shock will be felt. In some cases the systolic movement is of an undulatory character over the præcordia. With the retraction, the apex is noted to be displaced outward and the area of impulse is increased. The increase in area of impulse is due to the hypertrophy which always attends adhesion of the pericardium when it is universal. After the systole there is frequently felt a quick rebound known as the diastolic shock, which is said to be characteristic of pericardial adhesions. The area of cardiac dulness is increased usually upward, extending as high as the first interspace. The area of dulness is frequently not modified by respiration, that is, it is not lessened when the patient takes a full breath and the lungs expand over the præcordial region. This is particularly the case when there is pleuritis associated with pericarditis, a common association in the large majority of cases. On auscultation the signs vary. The sounds are due to hypertrophy or to dilatation; and it must not be forgotten that they frequently arise on account of pericardial adhesions. In the former condition the first and second sounds are accentuated; in the latter, a murmur may be heard at the apex, loud and systolic in time.

In pericardial adhesions, Friedreich's sign, *collapse* of the *cervical veins*, is seen. The collapse of the cervical veins takes place during the diastole of the heart. In addition the *pulsus paradoxus* is significant of the presence of pericardial adhesions, or rather of the dilatation that succeeds the adhesions. The pulse is small and feeble during

inspiration, assuming greater strength during the period of expiration. In pericardial adhesions the physical signs depend upon the condition of the heart muscle at the time of the examination. At first we have the physical signs of hypertrophy with retraction of the interspaces, particularly at the apex, or the space at the xiphoid cartilage. This is particularly the case in young subjects. In the later period of the disease the physical signs of dilatation arise, indicated by increase in transverse dulness, enfeeblement of impulse and of sounds, with the development of a murmur at the apex, undulation of the veins in the neck, and the pulsus paradoxus. The physical signs of associate pleurisy aid in the recognition of adherent pericardium. Diminution of the breath sounds, increase in the area of cardiac dulness, lessened fremitus in the neighborhood of the heart pointing to pleural thickening, are associate evidence. Sansom considers the presence of pulmonary tuberculosis of value, as pointing to the occurrence of pericardial adhesions, for the associate pleural adhesions are likely to be attended by tuberculous pericarditis.

The subjective symptoms of dilatation and hypertrophy of the heart are the symptoms of adherent pericardium, and are of the nature of the process, which is in excess at the time of the examination.

Mediastinal Pericarditis is a condition in which the pericardium is adherent and thickened, and, with the tissues of the anterior mediastinum, involved in a mass of fibrous inflammation. The symptoms attendant on this condition are those previously described—pulsus paradoxus, collapsing jugular veins during diastole, due to the dragging upon the innominate veins and cava by the fibrous adhesions, or to stretching and narrowing of the aortic arch by these adhesions. (See Fagge.)

Endocarditis.

Endocarditis may be acute or chronic. In either form it is usually secondary. The acute form is divided into the simple and so-called malignant or mycotic endocarditis.

Simple Endocarditis. Acute endocarditis rarely occurs primarily. It usually occurs secondarily to general morbid processes. The pathological antecedents are acute rheumatism, tonsillitis, whooping-cough, scarlet fever, rarely smallpox and typhoid fever. It is of common occurrence in pneumonia and tuberculosis. It is frequent in chorea. In the simple form it occurs in septic inflammations and in debilitating diseases, as cancer. It may occur in gout and develop in the course of Bright's disease.

Symptoms. The symptoms of simple endocarditis are scarcely observed during the early course of the disease. The process is latent, and there are no indications of cardiac disease. The physical signs alone tell of its presence. Unless these are sought for the disease is overlooked. The subjective symptoms are negative. In the course of rheumatism or chorea the patient may complain of palpitation of the heart; increased frequency and irregularity may be observed. At the same time the temperature may increase without increase in the rheumatic symptoms, the rise of temperature usually calling attention to the

cardiac complication. The rise is usually not marked, and may not assert itself during the severity of the disease.

Physical Signs. On examination a murmur is detected at one of the cardiac areas. The murmur is soft, low in pitch, and observes the rules as to transmission, depending upon the site of the murmur. Instead of a distinct murmur a roughening of the first sound alone may be heard. It must not be mistaken for the murmur which occurs at the apex in cardiac dilatation or for the murmur which takes place in the course of fevers, or the murmur at the aortic or pulmonary areas due to anæmia, which, by the way, rapidly ensues in rheumatism and other affections.

Malignant Endocarditis. This affection develops in the course of rheumatism in but few cases only. It is very rare in chorea. It differs from simple endocarditis by its rarity in the above affections. It occurs more frequently in pneumonia than in any other disease. It arises in the course of erysipelas, septicæmia, puerperal fever and gonorrhœa. It may occur in the course of dysentery. It is usually a streptococcus infection.

The Symptoms. The symptoms are due to (1) the local infectious inflammation, (2) to emboli, (3) the physical signs. The general symptoms due to the morbid specific process are septic in nature. Four groups of symptoms are seen : (1) There may be chills and fever, occurring in paroxysms daily or at intervals of two or three days, the course resembling that of pyæmia. With each chill and febrile rise there are profuse sweats. Rapid exhaustion ensues. The fever, instead of being distinctly intermittent, may be irregular in type. (2) As septic infection arises, a typhoid state, which is of frequent occurrence, sets in. The temperature is irregular; extreme prostration, low delirium, sordes, subsultus, and all the symptoms of that state arise. (3) Some cases are characterized by the relative absence of general symptoms. At least, they are not marked, and may be mildly febrile only. The physical examination shows the occurrence of the marked endocarditis, attended by slight fever. In this group there has usually been chronic heart disease preceding the affection. The moderate fever with the physical signs may continue over a long period of time. (4) In another class of cases the symptoms may be almost entirely cerebral, resembling cerebrospinal or basilar meningitis.

Diagnostic Features. Of the pronounced and constant symptoms that attend the course of malignant endocarditis we have: (1) the occurrence of fever ; (2) the occurrence of the heart symptoms ; (3) the occurrence of emboli. 1. The *fever* may be intermittent, remittent, or continuous. As previously noted, when the latter type is present the temperature is high and associated with the typhoid state. The petechial *rashes* and erythema are common, so that, as pointed out by Osler, the disease may resemble the eruptive fevers. The *sweating* is profuse, contributing to the profound exhaustion which usually ensues. A *diarrhœa* of septic character occurs when the fever is remittent or continuous. In a few rapidly fatal cases *jaundice* has occurred. 2. The heart symptoms may be latent entirely, both subjective and objective. Repeated examinations are necessary in some cases to determine the presence of a murmur or to decide whether a previously existing

organic lesion is the seat of an acute process. Variations in the character of the murmur from day to day may aid in determining this. In organic *heart disease* with dilatation and failure of compensation, irregular fever may occur, followed by embolic phenomena. The association of the two conditions points to the nature of the process. 3. The embolic phenomena are due to escape into the blood current of soft vegetations on the valves of the left heart (for the right heart is rarely affected), which are carried by the blood stream into distant points of the circulation. Emboli occur in the brain, producing aphasia or hemiplegia; they occur in the retina, causing some complaints as to vision, but are more particularly recognized by an ophthalmoscopic examination. They occur in the kidneys, on account of which bloody urine is passed with renal pain. In nearly all cases the spleen is the seat of embolism, and in some instances infarctions may take place in this organ alone. The spleen is always enlarged, and the development of the infarct may be attended with pain and increased tenderness on pressure. Emboli in the skin and mucous membranes present the most striking phenomena. The hemorrhages underneath the skin are minute, due to the infarcts. They are seen in the extremities, but may also be found in the trunk. They occur in the mucous membranes, as those of the mouth and tongue. They are seen in the bulbar conjunctivæ, and in the conjunctivæ of the lids.

Diagnosis. When embolic phenomena are present the diagnosis is made without much difficulty. The more pronounced general symptoms distinguish it from simple endocarditis. The temperature range, the septic and typhoid symptoms, belong to the malignant form. The more prolonged cases with moderately continuous fever, which occur without primary cause, as puerperal fever, are frequently confounded with typhoid fever. This is readily appreciated when the symptoms of the two are compared. In both there is fever of a continued type, with the symptoms of the typhoid state, including delirium. In both there are enlargement of the spleen, diarrhœa, and abdominal tenderness. In both there are infarctions, although these are extremely rare in typhoid fever, and occur late in the disease. In both there is progressive exhaustion. In endocarditis the onset may be more abrupt. The fever does not present the regularity of type that is seen in the development of typhoid. In endocarditis there is more oppression and dyspnœa early in the course of the disease than in typhoid fever. The diazo reaction is not found in typhoid fever before the fifth day, but rarely, if ever, in endocarditis. The results of bacteriological examination distinguish the two affections. This ought to be of value in endocarditis, because the process is usually due to a staphylococcus or streptococcus infection; either micro-organism may be found in any suppurations which may possibly be present.

Malignant endocarditis must be distinguished from cerebro-spinal fever and from smallpox of a hemorrhagic form. Reliance must be placed upon the local cardiac symptoms and physical signs, and the preponderance of these over the other symptoms. Of course the occurrence of an epidemic of either and a history of exposure are of service in the

distinction of the diseases. Examination of the blood excludes the forms of malaria which formerly were mistaken for endocarditis.

Chronic Endocarditis. Chronic endocarditis may follow the acute form or develop in the course of atheroma or of endarteritis due to alcoholism, the poison of syphilis or of gout. With endarteritis the endocardial change may be part of the general degenerative changes which occur in the aging process. It may be of dynamic origin, often following prolonged heavy muscular exertion, on account of which the valves, particularly at the aortic orifice, are put upon a strain. The process is slow and insidious, and leads to the changes in the valve segments which constitute chronic valvular disease.

Symptoms. The symptoms of chronic, or sclerotic, endocarditis are the symptoms of chronic valvular disease. Insufficiency or obstruction, or both combined, take place at the affected valve orifice. The outflow of blood is retarded in obstruction. Backward flow, or regurgitation, takes place in insufficiency in the opposite direction from the normal blood current. When there is obstruction hypertrophy usually takes place to meet it. If the obstruction is moderate, and the person remains in good health, the hypertrophy is sufficient to overcome the obstruction. In this manner the effect of the valve lesion is compensated. On the other hand, when blood is permitted to flow by regurgitation backward into the cavity, and hence opposite to its usual course, it meets at the same time blood flowing to this cavity in the normal direction, and the result is overdistention, or overfilling, of the cavity. Dilatation ensues, and may persist. If the regurgitation takes place suddenly the dilatation continues; if gradually, as in chronic endocarditis, the dilatation is attended with hypertrophy. Thus, when there is regurgitation from the left ventricle into the left auricle, on account of incompetency at the mitral orifice, the auricle becomes overdistended with blood, for at the same time the chamber is filling with blood from the pulmonary veins. This overdistention can only be overcome by some hypertrophy. When the latter is not sufficient, backing of the blood upon the pulmonary circulation takes place, with the consequences hereafter to be mentioned.

The symptoms of chronic endocarditis are latent if the lesions are compensated for; if not, symptoms of failure in compensation or *dilatation* of the heart arise. The physical signs are those of *chronic valvulitis*. The character of the signs is dependent upon the lesion of the valve that is the seat of disease.

Myocarditis.

Myocarditis may be acute or chronic. General myocarditis is always acute. The local form may be acute or chronic, depending upon the degree of the primary cause. The entire muscle or a portion only may be affected. The local variety is usually due to a thrombus in the terminal endings of the coronary artery, which cuts off the blood supply. The changes are those of myocarditis, to which may be added necrosis of small areas and the development of aneurism. *Etiology.* Pathological antecedents of acute general myocarditis are the fevers,

particularly typhoid fever and typhus fever, pneumonia, diphtheria, and septic fevers generally. Chronic myocarditis is usually associated with atheroma, one of the causes of which obtains and occurs in the later stages of Bright's disease. (See Atheroma.) The result of myocarditis, when acute, is the occurrence of dilatation of the heart or the development of fatty heart, or of aneurism of the heart. Chronic myocarditis is followed by the fatty heart, by dilatation, by the so-called fibroid heart or fibrous myocarditis, by aneurism. The above facts in ætiology are important in diagnosis.

Symptoms. The symptoms of *acute* myocarditis are vague. In the course of one of the above-mentioned diseases the patient may complain of some oppression in the præcordia and suffer from dyspnœa; attacks from syncope may occur, and sighing may be frequent. The pulse becomes more rapid and weak, but is usually not irregular. The circulation is much depressed, the hands may be cold, the face pallid. These symptoms may point simply to the extreme exhaustion that follows fever, although there is no doubt that some myocarditis exists in all cases, particularly if high temperature is present in the course of the fever. In many cases no symptoms referable to the heart are complained of, death taking place suddenly, in the course of the disease or after it has spent its force, on account of acute dilatation or cardiac paralysis. This is particularly the case in pneumonia and in the course of diphtheria. In the latter affection the sudden supervention of cardiac symptoms, dyspnœa, cyanosis, and cold extremities, may be due to paralysis of the heart. *Physical Signs.* Enfeeblement of the heart sounds, with sometimes increased accentuation of the mitral first sound, is observed. The impulse and apex beat are absent or scarcely detected at all. If acute dilatation supervenes the area of dulness may be increased.

The symptoms of *chronic myocarditis* are obscure and indefinite, and in the majority of cases depend upon the secondary changes that have taken place in the heart muscle. If there is *atrophy* of the fibroid heart, the pulse is feeble, slow, and irregular. It may be as infrequent as thirty or forty beats to the minute. Irregularity is not necessarily present, but intermittency is of frequent occurrence. Dyspnœa is complained of, aggravated by exertion. Attacks of angina pectoris are liable to occur. The symptoms of dilatation of the heart may ensue later, with the occurrence of œdema, cyanosis, and congestions. In *fatty degeneration* of the heart the pulse is increased in frequency, there is cardiac irregularity, palpitation, and the occurrence of dyspnœa. These, however, are also the symptoms of dilatation, which usually succeeds the degeneration. The heart sounds are weak. If dilatation has set in a murmur is heard at the apex, with galloping rhythm of the heart. In fatty degeneration there is a tendency to syncope, and slowing of the pulse rate. Shortness of breath on exertion may occur. Cardiac asthma occurs at night, and sighing and yawning are of frequent occurrence during the day. Sleeping is usually poor. The cerebral functions are in abeyance more or less, the action of the mind sluggish; the patient may have delusions or become maniacal. Cheyne-Stokes breathing was formerly thought to be of diagnostic significance.

Chronic myocarditis must be distinguished from *fatty overgrowth* of the heart. This cardiac change is frequently seen in brewers and publicans, and is usually associated with obesity. The pulse may be feeble, the heart sounds weak and muffled. The patients are subject to attacks of asthma, and frequently have bronchitis and emphysema. Vertigo is of common occurrence. Death may occur during syncope.

Aneurism of the Heart.

Aneurism of the valves following endocarditis cannot be recognized during life. Aneurism of the walls usually results from chronic myocarditis. The aneurism develops at the apex in the left ventricle. The symptoms are indefinite. In rare cases there has been noted marked bulging in the region of the apex, and a tumor is made out which may perforate the chest wall. A projection beyond the normal line of cardiac dulness may be made out by stethoscopic percussion. The symptoms are those of myocarditis and of dilatation of the heart.

Rupture of the heart is one of the causes of sudden death often without previous symptoms. The accident takes place during exertion. Quain collected one hundred cases, in seventy-one of which death took place without previous warning. In other instances there was a sense of anguish and suffocation in the cardiac region. The physical signs of slowly developing pericardial effusion may be ascertained if the leakage from rupture is slow in progress.

Chronic Valvular Disease.

Valvular disease includes valvulitis and valvular incompetency, and is recognized by (1) symptoms due to the effect of the lesion upon the general circulation; (2) by the physical signs of a valve lesion; and (3) by the physical signs of alterations in the heart muscle, which take place on account of the valve affection. In valvular disease there is either obstruction or regurgitation at the orifice which is affected. The former is always due to endocarditis in some form, the latter may be due to endocarditis, or to inability of the valve segments to close the orifice, which has become abnormally enlarged. The lesions cause disturbance of the flow of blood through the heart. The symptoms differ at different periods of the course of the disease. When the disturbance in the circulation is overcome by hypertrophy, and compensation is fully established, there are no symptoms, but only the physical signs of the valve lesion and of hypertrophy or dilatation. When compensation fails or is broken the symptoms of dilatation of the heart arise. In the consideration of valvular disease it is more profitable to take up the symptoms of each valve lesion, bearing in mind that two or more of the valves may be diseased at the same time, or that both obstruction and regurgitation may be present at the same time at the same valve orifice.

Aortic Regurgitation, Insufficiency or Incompetency. This may exist for a long period of time without presenting any symptoms. It occurs more frequently in men than in women, and is more

common in the later periods of life. It may be due to congenital malformation, to acute endocarditis, or, as is most frequently the case, to chronic endocarditis, and particularly that form which follows strain or undue exertion; alcoholism and syphilis are also frequent antecedents of this condition. In rare cases it follows rupture of the valves. Relative insufficiency or incompetency is of very rare occurrence. Insufficiency is frequently combined with obstruction.

On account of insufficiency, or regurgitation, at the aortic orifice the blood falls directly into the left ventricle during the diastole. There is first a relative diminution in the amount of blood in the artery; and second, an increased amount of blood in the ventricle, because the regurgitated column of blood meets the blood from the auricle which is filling the chamber at the same time. Dilatation of the left ventricle ensues, followed by hypertrophy. Dilated hypertrophy thus arises. The heart becomes enormously enlarged. This is one of the conditions in which enormous enlargement takes place—so-called *cor bovinum*. Occurring at the period of life and from the causes above mentioned, more or less sclerosis of the arteries attends this valve lesion.

The General Symptoms. They may be entirely negative as long as perfect compensation exists. This is particularly the case if there is but little general arterial sclerosis. Coincident lesions of other valves tend to break the compensation. The earlier symptoms are those due to arterial anæmia, particularly anæmia of the brain. There are headache, dizziness, and flashes of light before the eyes. The patient is of an anæmic appearance, and soon begins to suffer from shortness of breath. This at first develops upon slight exertion. Palpitation and oppression about the chest are complained of, readily excited by undue exertion. Pain is a common symptom. It may be complained of in the region of the præcordia, is of a dull aching character, and may radiate over these regions to the neck and down the arms, particularly of the left side. The anginoid pains may be followed by attacks of true angina pectoris. The latter are more common in aortic regurgitation than in any other valve lesion.

As compensation fails the dyspnœa increases. It takes place at night and compels the patient to sleep in a semi-erect posture in bed. Stases occur. Congestion of the lungs takes place, on account of which we have cough. Hemorrhage occurs, but not as frequently as in mitral disease. Œdema of the feet sets in, but general anasarca is not common. Œdema of the feet may be due to anæmia.

In aortic insufficiency sudden death is of common occurrence. This may take place at night during an attack of dyspnœa, or occur suddenly upon the slightest exertion, such as straining at stool, or the ascending of a height, or walking more quickly than usual.

The Physical Signs of Aortic Regurgitation. Inspection. The apex beat is downward, outward, and to the left. It may be as low as the seventh interspace, and as far out as the anterior axillary line. The area of cardiac impulse is increased. It occupies the whole præcordia, and heaving of the lower half of the chest may be seen. In young subjects there is præcordial bulging.

Palpation. The impulse is strong and heaving. After compensation

fails it is indefinite and wavy. A thrill, diastolic in time, may be felt with the hand placed about the middle of the sternum.

Percussion. The area of dulness is increased. The extent is greater than that of any other valve lesion, and is more particularly downward, and to the left.

Auscultation. At the second costal cartilage on the right a murmur is heard, diastolic in time. This may be its seat of maximum intensity. It is transmitted along the course of the sternum toward the apex. In some instances the maximum of intensity is greatest at the fourth left costal cartilage, or even at the apex. The second sound is absent in the large majority of cases. In some instances, however, both murmur and second sound may be heard at the same time. Other murmurs also are heard in aortic regurgitation, not always due to disease of the aortic valves :

1. A systolic murmur at the second costal cartilage on the right, transmitted into the vessels of the neck, short, rough, and high in pitch. It is due to roughening of the valve segments or to atheroma of the aorta.

2. A murmur at the apex, rumbling in character, localized to this area, usually presystolic in time. It is the murmur described by Flint, who attributes it to flapping of the mitral segments, which during diastole are not forced back against the heart wall. They remain in the blood current and produce relative narrowing.

3. A systolic murmur in the mitral area, low in pitch, due to dilatation. This occurs when failure in compensation takes places.

Examination of the Arteries. Pulsation is more common in the peripheral vessels in aortic regurgitation than in any other valve lesion. The carotids throb, the temporals pulsate, the brachial and radial arteries are conspicuous. Pulsation of the retinal arteries is seen by the ophthalmoscope, and has often led to the recognition of the disease by the ophthalmologist who had been consulted for other conditions. The pulsation is of a jerking character; in the neck it may simulate the pulsation of an aneurism. The aorta can be seen and felt at the suprasternal notch. The abdominal aorta pulsates vigorously in the epigastrium. On *auscultation* of the arteries double murmurs may be heard in the carotids and subclavians, and in rare instances they are present in the femorals. (See Pulse.)

The Capillary Pulse. This is seen beneath the finger-nails, or on the surface of the skin, as the forehead, when a line is drawn across it. The hyperæmia produced on either side of the line alternately becomes red and then pale. Capillary pulse also occurs in anæmia, and at times in neurasthenia.

The Pulse. The pulse is significant in aortic regurgitation. The so-called water-hammer, or Corrigan's pulse, is observed. The pulse is quick and jerking, and after striking the finger immediately recedes. It is most marked when the arm is held up.

Aortic Obstruction. Disease at the aortic orifice causing obstruction to the flow of blood is rare. It occurs in the aged and with atheroma of the arteries. It causes some diminution in the amount of blood in the peripheral circulation, which causes poor nutrition and the development of anæmia.

Symptoms. Anæmia develops, and embolic phenomena may occur later. The symptoms may be latent until the occurrence of the latter accident.

On account of the position of the aortic valve, embolic symptoms are not uncommon. The emboli are distributed throughout the arterial circuit, and may take place in the brain, kidneys, or spleen. When the obstruction is pronounced, there is lessened supply of blood in the arteries. Cerebral anæmia here takes place, causing dizziness and fainting. Sleep is more disturbed than in other valve affections, because of the cerebral anæmia. Palpitation and cardiac pain occur, but are not so common as in aortic regurgitation. When compensation fails, dilatation of the left ventricle ensues, followed by pulmonary congestion and stases in the systemic circulation.

The Physical Signs. There is hypertrophy of the left ventricle. *Inspection.* The apex beat is displaced downward and outward. The impulse is strong during the period of hypertrophy. When compensation fails the physical signs of dilatation ensue. In many cases, from the very first, there may be considerable hypertrophy without visible impulse, because of associate emphysema, which is common in old men.

Palpation. At the base of the heart, and in the aortic area, a thrill, systolic in time, may be felt. When present, it is usually very distinct, and is transmitted along the course of the vessels. The impulse is slow and heaving, as in hypertrophy. In dilatation it is feeble and indistinct.

Percussion. The area of dulness is increased, in the earlier stages, to the left and downward. After compensation is broken, dilatation with increased dulness ensues.

Auscultation. A murmur of maximum intensity at the second costal cartilage to the right, systolic in time, transmitted in the course of the bloodvessels, is heard. It is usually harsh and loud, and may be musical. As the heart weakens, the degree of loudness of the murmur lessens and its roughening disappears. It becomes soft and low in pitch. The second sound, if there is no regurgitation, is muffled or may be absent. The *pulse* is small and regular. The tension is usually increased.

Diagnosis. A systolic murmur at the aortic orifice may be due to aortic obstruction, to atheroma or dilatation of the aorta, ulcerative aortitis, or to anæmia. The murmur of aortic stenosis is distinguished from the others by its character, by the presence of thrill, by the character of the pulse, and by its association with hypertrophy of the left ventricle. A murmur due to atheroma of the aorta, particularly in the course of renal disease, is also associated with hypertrophy of the left ventricle, and the distinction is often difficult or impossible. The slowness of the pulse is more characteristic of aortic obstruction. The murmur of anæmia is softer and low in pitch. There is no thrill, and the left ventricle is not hypertrophied. The anæmic murmurs may be heard elsewhere. In atheroma the second sound is usually accentuated, and in anæmia it is also intensified.

Mitral Incompetency or Regurgitation. The regurgitation may be due to disease of the valves from previous endocarditis, which is

usually of rheumatic origin, or to inability of the segments to close the orifice, enlarged on account of dilatation of the cavities. The latter occurs in dilatation of the left ventricle under all circumstances, and in the weakening of the muscle that occurs in fevers and in anæmia. It is thus seen that the murmur of mitral insufficiency is one of the most commonly observed of all valve murmurs. It must not be forgotten that insufficiency from disease of the valves and from disease of the muscles must, if possible, be distinguished from each other. The history of the case is usually essential in determining the diagnosis.

Disease at the mitral orifice producing insufficiency has more serious effects upon the pulmonic and arterial circulation than disease at any of the other orifices. These effects must be understood in order to appreciate the symptoms of mitral incompetency. They are as follows: 1. With each systolic contraction the blood flows back, on account of the insufficiency, to the auricle, where it soon meets a volume of blood coming from the lungs. The combined volumes of blood overdistend the auricle. Dilatation ensues, and because of increased work to get rid of the increased contents, hypertrophy follows. Dilated hypertrophy of the left auricle is the first effect. 2. A larger amount of blood is forced, as a result of the above, from the auricle into the left ventricle; dilatation and subsequent hypertrophy of this chamber follow, to remove the fluid. 3. On account of the overdistended auricle, the pulmonary veins are not fully emptied during the diastole of that chamber. The veins are therefore engorged and interfere with the flow of blood through the pulmonary circuit. In consequence of the back-flowing of blood, the vessels in the pulmonary circuit are dilated and overdistended with blood. The right ventricle is compelled to act more vigorously, and even then cannot empty itself freely. Dilatation and hypertrophy of the right ventricle ensue. 4. This causes obstruction of the flow of blood from the right auricle to the right ventricle; dilatation and hypertrophy of its chambers follow. If perfect compensation ensues through hypertrophy of both ventricles, engorgement in the lungs may not be observed. Moreover, the left ventricle is allowed to send out sufficient blood to supply the wants of the system. This compensation may continue for years. If it fails, either from increase in the valve lesion, or incompetency, or from weakening of the muscle, a normal amount of blood is not distributed throughout the aortic area, but is thrown back upon (1) the left auricle; (2) the pulmonary circulation; (3) the right heart; and, finally, the systemic veins. For a time the pulmonary circuit will alone be engorged, subsequently the systemic veins become congested because of dilatation of the right auricle and incompetency of the tricuspid valves. We then have the occurrence of the secondary effects of stases upon the various organs of the body, with cyanotic induration and the development of dropsies. Mitral incompetency without disease of the valves is of frequent occurrence in emphysema of the lungs and in Bright's disease, and is a condition which always attends hypertrophy and dilatation, or may take place from various causes (see Hypertrophy and Dilatation).

Symptoms. As to the general symptoms: In a large number of cases perfect compensation may continue throughout a long period of time.

No subjective symptoms arise nor are there symptoms due to dilatation. If compensation is not perfectly effected from the first, or is broken suddenly or gradually, the symptoms of dilatation arise.

In patients in whom compensation remains fairly good we have the characteristic appearances of a subject of heart disease. It is to this class of patients that the general descriptions of heart disease apply. The face is pale and pinched, the lips and ears dusky, the capillaries of the cheeks enlarged, the finger-nails clubbed, particularly in children; shortness of breath on exertion may be the only symptom complained of, and this may exist for years. Patients, however, are liable to attacks of bronchitis, and may have attacks of pulmonary hemorrhage. Palpitation of the heart may occur in this as in other forms of heart disease, and from the same cause.

When the compensation is broken symptoms referable to the heart and to engorgement of systemic and pulmonary veins occur. Of the former palpitation with a sense of oppression is the most common; pain is rare.

Venous engorgement leads to congestions, cyanosis, and dropsies. The lungs are the first to be congested. Dyspnœa becomes constant as well as aggravated by exertion. Cough is present, excited by exertion or speaking. With the cough there is bloody expectoration. Cyanosis occurs. Congestion of other organs follows. The liver is enlarged; obstruction in the portal area is prominent; chronic gastritis or gastro-intestinal catarrh ensues. The spleen is enlarged; ascites develops, and hemorrhoids and congestion in the rest of the portal area are seen. The kidneys are congested; the urine is scanty, albuminous, and contains casts and blood corpuscles. At the same time that the internal viscera are congested dropsies take place, beginning in the feet and extending to the rest of the body. Dropsy may have been present in the feet before symptoms of portal congestion ensued. The patient may be relieved and compensation continue good for a long time. Frequent attacks of dilatation of this character may take place, their recurrence being due to lack of care in hygienic matters, or failure in health from other causes. Finally, however, the compensation cannot be restored; the stases persist; the dropsies become more marked, and the symptoms of chronic cyanotic induration and secondary sclerosis of the internal organs follow. It must not be forgotten that this is the chief form of organic heart disease seen in children.

Physical Signs. On *inspection*, the prærcordial area is prominent; the apex beat is displaced to the left and downward, rarely below the sixth interspace. It may extend to the anterior axillary line. The cervical veins pulsate and are distended. The area of impulse is increased.

Palpation. The character of the impulse depends upon the stage of the disease at which the case is examined. At the time of full compensation it is strong and even. When this is broken it is feeble and diffuse. A thrill is extremely rare.

Percussion. The area of dulness is increased to the left. The transverse width of the heart is much increased because of dilatation of both chambers. The area extends beyond the right margin of the

sternum to the extent of an inch or more and to the left as far as the mid-clavicular line; sometimes to the anterior axillary line.

Auscultation. At the apex, *the mitral area*, a murmur is heard. The point of maximum intensity is in this region. It is systolic in time; it may replace the first sound entirely. It may be soft and low in pitch, or rough, high in pitch, even musical in character. It is transmitted to the axilla and the angle of the scapula. In some instances it may be heard loudest along the left border of the sternum. The pulmonary second sound is accentuated; the accentuation is loudest in the pulmonary area at the third left interspace. It may be heard very loud over the right ventricle between the parasternal line and the left edge of the sternum. The murmur of mitral insufficiency is modified by the position of the patient and intensified after exertion. It may be present when the patient is lying down, and disappear in an erect posture. It may disappear when the patient is quiet and return after exertion. Other murmurs are sometimes heard:

1. A presystolic murmur, soft or rumbling. 2. When dilatation ensues, a low-pitched systolic murmur is heard at the ensiform cartilage and at the lower left border of the sternum. It is due to tricuspid regurgitation.

The Bloodvessels. The amount of blood in the arteries is diminished. There is notable absence of pulsation of the arteries. The pulse at first is full and regular. It is notably small in volume and soft. As soon as failure of compensation takes place the pulse becomes irregular. The irregularity may be that of time as well as volume.

Of special *diagnostic significance* we have the position of the murmur and the direction of its transmission; accentuation of the pulmonary second sound; enlargement of the transverse diameter of the heart due to dilatation of both ventricles.

Diagnosis. This is usually easy if the physical signs are sought for. Very often patients are treated for the symptoms that arise from congestion of the viscera without an examination of the heart having been made. We have often seen chronic gastritis or gastro-intestinal catarrh due to mitral insufficiency not relieved because the primary lesion had not been ascertained. In the same way cardiac cough or dyspnœa may be overlooked. It is important in the diagnosis to determine if possible the nature of the insufficiency, whether it is due to disease of the valves or to incompetency. As previously mentioned, the history is possibly the only means by which a diagnosis can be made. If a mitral murmur ensues in old people in whom there has been physical cause for the development of dilatation and hypertrophy, as in emphysema or arteriosclerosis, it is usually due to incompetency of the valve leaflets to close the orifice. It must not be forgotten that the mitral area is the seat of a number of murmurs due to various causes. (See Auscultation.)

Mitral Stenosis. Obstruction to the flow of blood from the auricle to the ventricle is the result of endocarditis, and particularly the endocarditis of early life. It is of much more frequent occurrence in women, in contradistinction to aortic disease. As intimated, it is much more frequent in young adults and children, because its ætiological factors, rheumatism and chorea, are more prevalent.

On account of the obstruction of the orifice changes ensue in the auricle. These changes depend in a measure upon the nature of the lesion. In the so-called buttonhole contraction they are very marked. The orifice may be so obliterated in rare cases as to admit only a small probe. Dilatation and hypertrophy of the left auricle ensue if the valve changes take place gradually. The walls of the auricle are thickened to three or four times their natural size. On account of the dilatation of this auricle the outflow from the pulmonary veins is impeded, which in turn obstructs the circulation of blood through the lungs. As a consequence dilatation and hypertrophy of the right ventricle occur. As a result of this we have, later on, the occurrence of relative incompetency at the tricupsid orifice with engorgement of the systemic veins. The left ventricle does not take part in any changes. It retains its normal size, but it may look small in comparison with the right ventricle.

Symptoms. If hypertrophy of the right ventricle ensues, the compensation may be sufficient to prevent the occurrence of symptoms for many years. The disease may exist for a number of years without discomfort to the patient. Because of its rheumatic origin endocarditis may recur, particularly as the subjects are young, and hence may cause danger. If fresh endocarditis occurs embolic symptoms are likely to take place. These may take place in the brain particularly, causing hemiplegia or aphasia. When failure of compensation takes place the symptoms described in mitral incompetency arise. They are the symptoms of dilatation of the heart. These symptoms may recur frequently during a long period of years.

Dropsy, however, is not of as common occurrence as in mitral regurgitation. Visceral stases are common when compensation fails, and in many we find enlargement of the liver continuing over a long period. Ascites may in rare cases be the only manifestation of mitral obstruction.

The Physical Signs of Mitral Obstruction are more striking and pronouncedly diagnostic of the lesion than the physical signs of any other form of organic heart disease. As the disease develops in children the local deformities are more marked than in adults.

Inspection. For the latter reason præcordial bulging is more prominent. Because the right ventricle is hypertrophied, the sternum and the fifth and sixth costal cartilages protrude. As the left ventricle is small, the apex beat is not easily found. It is not usually dislocated, certainly not beyond the mid-clavicular line. The impulse is not marked at the apex. In the third and fourth interspaces a visible impulse is seen along the margin of the sternum. After dilatation, the extent of impulse diminishes and the veins of the neck become engorged, the blood regurgitating into them during the systole. *Palpation.* In the large majority of cases a distinct fremitus or thrill is felt—more marked in the fourth or fifth interspace, inside of the nipple. It is usually localized to a small area, is increased during expiration, and is of a twisting, grating or grinding character, usually rough. It is made up of a series of small shocks increasing in intensity, culminating in a sudden, sharp shock, which occurs at the time of the impulse. The thrill is pathog-

nomonic and may be present when other signs, as the murmur, are absent or indistinct. The cardiac impulse is felt strongest at the lower margin of the sternum and in the third and fourth interspaces, in some cases even in the second. It is due to enlarged and dilated right ventricle.

Percussion. The area of cardiac dulness is increased upward and to the right and left of the margin of the sternum. The extent of the increase of the area of dulness upward as high as the second rib sometimes is quite characteristic.

Auscultation. At the apex or just inside of the position of the apex beat, a murmur is heard, its point of maximum intensity distinctly localized to this situation. It is not transmitted. It is of a churning and grinding character, or vibratory and purring. It is usually high in pitch and rough. It occurs synchronously with the thrill and terminates with a loud shock that is heard simultaneously with the first sound. It is therefore presystolic in time. As has been said of the thrill, so of this murmur it may be said that it is the only murmur that is pathognomonic of a special lesion. It indicates narrowing of the mitral orifice. The only exception in which this lesion is absent, and yet the murmur is present, is in the class of cases described by Flint, referred to in the the section on aortic regurgitation.

The presystolic murmur may occupy the entire period of the diastole. In the large majority of cases it occurs in the latter half only, during which the auricular systole occurs. In some instances it is heard in the middle of the diastole.

Associate Murmurs. 1. At the same time a systolic murmur may be heard at the apex, soft, and low in pitch. It may be transmitted into the axilla. It is usually due to associate mitral regurgitation. 2. At the lower portion of the sternum a systolic murmur is heard, due to dilatation and incompetency at the tricuspid orifice. Murmurs in the aortic region are not usually heard. The second sound at the pulmonary orifice is usually heard accentuated. It is heard in the second and third interspaces along the left edge of the sternum; it may be heard at the apex. After compensation is broken other murmurs may be heard, and the presystolic murmur changes in character. It may disappear entirely and be replaced by a sharp first sound. The short, high-pitched systolic shock may continue, although the audible murmur disappears. It disappears, probably because the left auricle has become weakened. The tricuspid murmur continues during this period. The points of distinction are (1) the position of the murmur; (2) its localization; (3) its peculiar character; (4) the systolic shock which takes the place of the first sound; (5) the thrill; (6) the impulse and increased area of dulness upward; (7) accentuated pulmonary second sound.

Tricuspid Regurgitation or Incompetency. Structural disease at the tricuspid orifice is of extremely rare occurrence. Insufficiency is comparatively frequent, and is due to dilatation, with relative insufficiency of the valve orifice. It occurs secondarily to destructive lung diseases, as emphysema, and cirrhosis, and is secondary to regurgitation at the mitral orifice on account of which stases in the lungs have taken place.

The Symptoms. The symptoms have been detailed in speaking of the mitral valve affections. They are those of obstruction in the pulmonary circulation and engorgement of the systemic veins. On inspection, the physical signs of dilatation of the right heart are seen. An impulse in the epigastrium is noted. This is seen especially between the xiphoid cartilage and the left margin of the ribs. Pulsation to the right of the sternum may also be observed. There is also pulsation in the second and third intercostal spaces. The veins of the neck are also seen to pulsate. In addition to the wavy pulsation, regurgitation of the blood into the right auricle causes transmission of the pulse wave into the veins. The pulsation is systolic in time. It is more marked in the right jugular than in the left, and in the external than in the internal veins. With the pulsation, regurgitation is readily observed by emptying the external vein. Place the finger with firm pressure on the vein just above the clavicle, move it along the course of the vein in the direction of the inferior maxillary bone. The vein is thus emptied of blood, and with each systole of the heart it will be seen to fill up from below in rhythmical pulsation. In addition to the pulsation of the veins they are increased in size. This is more noticeable during the act of coughing or when the patient holds the breath in full inspiration. In rare instances the pulsation is transmitted to the subclavian and axillary veins.

The regurgitant pulsation is transmitted to the inferior vena cava as well as to the ascending. The hepatic veins also distend during the systole. So-called pulsation of the liver is produced. With the one hand on the fifth and sixth costal cartilages and the other over the liver in the axillary region rhythmical expansile pulsation may be recognized. It is not of common occurrence, but is absolutely diagnostic of regurgitation at the tricuspid orifice.

Palpation. By palpation the above conditions are determined. The impulse over the lower sternum and in the epigastrium is noted to be forcible.

Percussion. The area of cardiac dulness is increased transversely and upward as described in mitral stenosis. It extends often far beyond the right edge of the sternum.

Auscultation. At the xiphoid cartilage or the lower end of the sternum a murmur is heard. It is systolic in time, usually low in pitch and is heard loud to the left of the sternum, within an inch of the apex, and to the right of the sternum and the outer limits of percussion dulness. Other murmurs are heard due to the primary organic disease. The pulmonary second sound is accentuated.

Tricuspid Stenosis. Stenosis at this valve orifice is generally of congenital origin. In rare instances it may be secondary to lesions in the left heart. It is accompanied by dilatation of the right auricle. The physical signs are the same as in stenosis at the mitral orifice, except the alteration in their position. In some instances a presystolic thrill has been observed and with it a presystolic murmur at the lower end of the sternum or toward the right of it. The area of dulness is increased as in right-sided dilatation. Cyanosis is a most prominent symptom and may be very intense.

Disease of the Pulmonary Valve. Diseases of the pulmonary valve are extremely rare and are almost always congenital.

Pulmonary Stenosis. In stenosis of the pulmonary valve a systolic murmur and thrill to the left of the sternum in the second interspace are detected. The murmur is not transmitted to the vessels of the neck. The pulmonary second sound is weak. The effect on the heart is the production of right-sided hypertrophy.

Pulmonary Insufficiency. The physical signs are due to regurgitation into the right ventricle. The maximum intensity of the murmur is in the second pulmonary interspace, and it is transmitted down the sternum. It cannot be told from aortic regurgitation, except by the pulse.

Combined Valvular Lesions. It must not be forgotten that disease causing both obstruction and regurgitation can take place at the same time, or that two or more valves may be the seat of disease in the same individual. It is not impossible, for instance, to have aortic obstruction and regurgitation, mitral obstruction and regurgitation, and tricuspid regurgitation. Aortic obstruction or insufficiency is frequently combined with mitral insufficiency. Aortic and mitral insufficiency occur together most frequently in children; aortic obstruction and mitral obstruction in adults.

When more than one valve is diseased the detection of the various lesions is based upon the time of the murmurs, the position of their maximum intensity, and the direction of their transmission. Students often experience difficulty here. A systolic murmur may be heard in the aortic area and in the mitral area at the same time. If it is observed that each progressively weakens as the stethoscope is moved toward the middle of the præcordial area it may be inferred that the murmur, systolic in time, is due to two lesions. As previously intimated, the direction of the transmission of the murmur further aids in the diagnosis.

Enlargement of the Heart.

Enlargement of the heart is due to *hypertrophy* or to *dilatation*. In hypertrophy there is increased thickness of the muscular walls. This may be general or limited to the walls of one chamber. Hypertrophy is further divided into simple hypertrophy, in which the cavity or cavities are of normal size, and eccentric hypertrophy, in which, with increase in the wall, there is enlargement of the cavities. This is hypertrophy with dilatation. The left ventricle is most frequently the seat of hypertrophy when one chamber is involved. The cause of hypertrophy is obstruction to the flow of blood; increased work is followed by increased size of the muscle. *General hypertrophy*, or *hypertrophy of the left ventricle*, occurs from diseases of the heart itself, or from affections of the bloodvessels.

A. *Diseases of the heart.* 1. Disease of the aortic valves. Hypertrophy of the left ventricle always follows. 2. Mitral regurgitation. 3. Pericardial adhesions. 4. Myocarditis of the fibrous variety. 5. Neuroses with overaction and frequent palpitation, as in exophthalmic goitre and from the effects of tea, tobacco, and alcohol. In pericardial

adhesions and myocarditis, hypertrophy arises because of the inability of the heart to do the work expected of it. There is no obstruction in the course of the vessels or at the orifices. The struggle to keep up causes the hypertrophy. In neuroses there is absence of obstruction, but the rapid action causes hypertrophy.

B. *Affections of the bloodvessels* which cause hypertrophy are: 1. General arterial sclerosis. 2. Increased arterial tension due to the contraction of the peripheral arteries, as in Bright's disease, and in toxæmias from lead, the poison of gout, and syphilis. 3. Increased blood-pressure from prolonged muscular exertion. 4. Narrowing of the aorta from external pressure and from congenital stenosis or the development of an aneurism.

Hypertrophy of the Right Ventricle. In hypertrophy of the right ventricle obstruction to the flow of blood throughout the pulmonary area is the causal condition. This occurs because of lesions of the mitral valve, causing pulmonary stasis; disease of the lungs, causing compression of the bloodvessels, as in emphysema or cirrhosis. It occurs if there is disease of the right heart with obstruction of the valves. In obstruction at the pulmonary orifice the right ventricle undergoes secondary hypertrophy.

Hypertrophy of the Auricles. Simple hypertrophy of the left auricle with dilatation develops in mitral stenosis. Hypertrophy of the right auricle occurs in right-sided dilatation with tricuspid regurgitation.

Symptoms. The symptoms of hypertrophy of the heart are *general* and *local*. The former are not common. They are due to increased force of the circulation through the brain, usually causing congestive headaches, with noises in the ears, flashes of light, and flushing of the face.

General symptoms arise in the course of hypertrophy of the left ventricle on account of the effect of the increased force upon the vascular system. In Bright's disease, for instance, or heightened arterial tension from other causes, endarteritis develops in the large vessels on account of the strain put upon them. This is seen particularly in the aorta and its divisions. Whether atheroma is primary or secondary, its presence with hypertrophy of the left ventricle indicates that rupture of the vessels somewhere in the periphery may take place. This occurs most frequently in the brain, causing apoplexy.

Locally the patient complains of fulness and discomfort, particularly marked when lying down on the left side. In the hypertrophy that accompanies the tobacco heart, or the irritable heart of soldiers, there may be some pain. The organ may be enormously enlarged without the patient complaining of discomfort about the heart. Palpitation is not of common occurrence except in neurasthenic subjects.

Physical Signs. If it has developed early in life, when the ribs are soft, the hypertrophy causes præcordial bulging. The intercostal spaces are widened and the area of impulse is much increased. The apex is changed in position. The hypertrophy of the left ventricle is downward and to the left, extending as far as the axilla.

Palpation. The impulse is forcible and heaving. If the ear is applied over the heart the head is visibly raised with each systole. The im-

pulse is slow. This slow, heaving impulse distinguishes it from the forcible impulse of dilated hypertrophy which is sudden and abrupt. Inspection is confirmed as to the position of the apex. In moderate hypertrophy the apex extends to the sixth interspace in the mid-clavicular line. In large-sized hypertrophy it may extend to the seventh interspace.

Percussion. The area of dulness is increased both upward and transversely. It may begin as high as the second interspace and extend two inches beyond the left mid-clavicular line and an inch beyond the right edge of the sternum transversely. In simple hypertrophy the area is ovoid.

Auscultation. When the valves are healthy, prolongations of the first sounds occur. They are also at times duller than in health. The dull, prolonged first sounds distinguish hypertrophy from dilatation, in which the same sounds are clear and sharp. The second sounds are clear and loud. The degree of accentuation depends upon the state of the peripheral arteries. If there is heightened tension the second sound may be reduplicated. If valvular disease is present the sounds are modified.

The Pulse. The frequency of the pulse is not modified. It is full, regular, and strong. The tension is increased. In dilated hypertrophy the pulse is full but soft, and more rapid than in simple hypertrophy. When failure of the heart takes place the pulse increases in frequency and becomes intermittent and irregular. When valve lesions are present the pulse is modified accordingly.

Hypertrophy of the Right Ventricle. Increased pulmonary tension from resistance in the pulmonary circulation may always be looked for. If there is complete compensation no symptoms are observed or only those of dyspnœa on extra exertion. Hypertrophy of this ventricle persists for a long period of time without the grave local changes in the heart or secondary changes in the peripheral vessels which occur in left ventricle hypertrophy. In dilated hypertrophy, when the dilatation is in excess, tricuspid regurgitation takes place, with the development of venous stases. Induration of the lungs succeeds the engorgement of the capillaries in dilated hypertrophy. When the dilatation is excessive pulmonary congestions and apoplexy are associated.

The Physical Signs of hypertrophy of the right ventricle have been partially referred to under the various valve affections. There is bulging of the lower part of the sternum and cartilages. The epigastric impulse in the angle between the ensiform cartilage and the ribs has been referred to. The impulse may be in the sixth interspace. The impulse is diffuse; it may extend upward as in mitral stenosis. Cardiac dulness is increased toward the right an inch or more beyond the border of the sternum. The heart sounds are not much changed unless there is dilatation. The tricuspid sound is clear and sharp when this occurs. The pulmonary second sound is accentuated and reduplication may take place. The radial pulse is small. If there is tricuspid regurgitation the physical signs that attend it are present.

Hypertrophy of the Left Auricle. This is present in mitral stenosis but cannot be determined by physical signs, save probably greater increase of dulness to the left of the sternum in the second and third

interspaces. *Hypertrophy of the right auricle* with dilatation occurs under the same circumstances as hypertrophy of the ventricle. It dilates more usually than the left auricle in left ventricle hypertrophy. There is increased area of dulness in the third and fourth interspaces; abnormal pulsation is sometimes observed in this situation before the systole, with the signs of tricupsid regurgitation.

Diagnosis. The forcible impulse that occurs in nervous palpitation of the heart must not be confounded with true hypertrophy, although it must not be forgotten that hypertrophy follows neurotic palpitation frequently, as in the smoker's heart, or in exophthalmic goitre. The enlargement must not be confounded with enlargement of the area of cardiac dulness in the præcordial region from other causes, such as pericardial effusion; aneurism and mediastinal tumor, pushing the heart against the chest wall; disease of the lungs, on account of which they are withdrawn from the surface of the heart, as in phthisis or chronic pleurisy; and displacement of the heart from pressure, as in effusion on the left side of the chest or disease below the diaphragm. The cause of hypertrophy should be ascertained, for when discovered it is a valuable aid in the diagnosis. It must not be forgotten that emphysema of the lung may mask a considerable hypertrophy of the heart by causing diminution of the area of dulness.

Dilatation of the Heart. Enlargement due to dilatation of the heart is common. The condition usually succeeds hypertrophy. Thickening of the muscles attends dilatation of the cavities, as in dilated or eccentric hypertrophy. The dilatation occurs because of increased pressure within the cavities or because of weakening of the heart walls, the pressure within being normal.

1. Increased pressure within the walls is due to an increased amount of blood within the chamber from regurgitation or an obstacle to the outward flow of blood. Simple hypertrophy occurs first in many cases; in others, hypertrophy with dilatation; in not a few, dilatation at once takes place. In dilatation the chamber does not empty itself during the systole. It is seen physiologically after the exertion of ascending a great height. It may remain within the bounds of physiological action. The dilatation is attended by increased epigastric pulsation, and sometimes increase in cardiac dulness. The tricuspid valves temporarily become incompetent, owing to their safety-valve action. It may continue after the acute strain, the heart always showing symptoms of the condition, or it may disappear entirely. The excessive dilatation that sometimes follows results in heart strain, with the cardiac distress of which dyspnœa is associated. Acute dilatation from overdistention and paralysis of the heart occurs (see Symptoms). Dilatation occurs in all forms of heart lesions which have been previously described. The most typical form occurs in aortic regurgitation, when the left ventricle becomes the seat of dilatation, and in mitral regurgitation when the left auricle becomes the seat of dilatation.

2. Disease of the heart walls lessening the resisting power precedes dilatation, the normal pressure within the cavities being maintained. In the myocarditis that occurs in the course of fevers, acute dilatation may ensue. It occurs in scarlatinal dropsy, typhoid fever, rheumatic fever,

and erysipelas. The heart muscle changes in acute endo- and pericarditis, on account of which dilatation may ensue. In anæmia and chlorosis the same process may take place. In chronic myocarditis, dilatation takes place at the apex. When pericardial adhesions are present, the fibrous overgrowth invades the interstices of the myocardium, weakening thereby the heart muscle. Dilatation may follow.

Symptoms. The symptoms of dilatation are the reverse of hypertrophy. When the latter fails, the blood is not expelled from the chambers in the systole, so that with the blood that accumulates in the diastole the chamber is overdistended. Weakening of the muscle aids further in the development of dilatation. As soon as dilatation becomes permanent, incompetency of the valves takes place. In obstructive heart disease, the left side is first affected. It may be compensated for by hypertrophy of the right side. When this fails, venous engorgement and dropsy ensue. The symptoms have been described under chronic valvular disease. In *acute dilatation* there is a sudden occurrence of dyspnœa. Pain may be complained of in the heart. With the dyspnœa, the heart's action increases in frequency. The pulse is rapid, feeble, irregular, and may scarcely be felt at the wrist.

Physical Signs. Inspection. The apex is displaced to the left, but rarely downward, unless hypertrophy precedes the dilatation. The impulse is diffused and undulatory in appearance. The apex beat may be defined with extreme difficulty. It may be visible when the patient leans forward, yet not felt.

Palpation. With the diffused area of impulse, a quick apex beat may be felt—much weakened, however. If the right heart is dilated, the true apex cannot be felt because the right heart comes in apposition with the surface of the chest. The impulse is seen and felt then to the right or left of the xiphoid cartilage, and there is a wavy pulsation along the left edge of the sternum in the fourth, fifth, and sixth interspaces. If the dilatation is extreme, involving the right auricle, a pulsation at the third right interspace close to the sternum may be felt. Tricuspid regurgitation is then present.

Percussion. The area of dulness is increased in the same directions as found in hypertrophy if the two coexist. In general, it may be said the increase extends outward to the right or left, the direction corresponding to the ventricle affected. It is increased upward along the left edge of the sternum in left auricle dilatation. When the whole heart is dilated, the increase of dulness is in a transverse direction on both sides. The apex is rounded or square, not pointed as in hypertrophy. As dilatation occurs so frequently in emphysema of the lungs, the modification of the percussion sound must be remembered.

Auscultation. The systolic sounds are short and sharp. They are high-pitched and resemble the diastolic. The latter may become enfeebled when the dilatation becomes excessive. The right and left first sounds may differ somewhat in intensity, and reduplication may occur. The sounds may be obscured by murmurs. The murmurs are due to previous valve disease or to incompetency on account of dilatation. The action of the heart is irregular and intermittent. The pulse is correspondingly small. In dilatation the alteration of the rhythm is extreme. There may be

embryocardia or fœtal-heart rhythm, in which the first and second sounds are alike, and the long pause is shortened. More frequently we have galloping rhythm of the heart. It must not be forgotten that as dilatation ensues, murmurs of various valve lesions may disappear, particularly the murmur of mitral stenosis. On the other hand, in the earlier stages particularly, murmurs develop on account of incompetency at the auriculo-ventricular orifices, in addition to the primary organic murmur. These murmurs in turn may disappear, if the dilatation is controlled by careful treatment.

Congenital Heart Disease.

Cyanosis is the chief symptom of congenital heart disease. The term *blue disease* and *morbus cæruleus* are used as synonyms for this condition. The lividity appears in the first week of life. It may be general or confined to distant points of the circulation. In extreme grades the skin is almost purple. It may vary from time to time, and be intense on exertion. The external temperature is below normal. If the child remains quiet there may be no symptoms of dyspnœa; dyspnœa and cough occur if it is moved about, or on exertion when the child is older. The physical development is very poor, the mind is sluggish. Clubbing of the fingers and toes takes place to a high degree. The recognition of the condition in children is not difficult. If a murmur is found in a patient with cyanosis during the early weeks of life, it is due to congenital heart disease. The murmur is usually systolic in time. Hypertrophy occurs in a number of cases. In some instances the murmur is absent.

Diseases of the Arteries.

Arterial Sclerosis or Arterio-capillary Fibrosis. This occurs as the result of wear and tear of life and as the accompaniment of age. The time of its onset depends upon the quality of the arterial tissue which the individual derived by heredity, and upon the amount of wear and tear. It may occur early in life, and entire families may show this tendency. Very frequently the sclerosis develops from intoxications of the system, on account of which persistent spasm of the small vessels is set up; or blood of an impaired quality is passed with greater difficulty through the capillaries, as was taught by Bright. The blood tension is raised thereby. The poison of alcohol, of lead, of gout, and of syphilis leads to this condition. The poison of syphilis and of gout may set up directly an inflammation and degeneration of the arteries. In renal disease, arterial sclerosis is of common occurrence. The relation to the renal lesion differs. It may be primary or secondary. When primary, the morbid cause operates upon the kidneys as well as the arteries. When secondary, a morbid poison is retained within the system by the diseased kidneys, the action of which is such as to cause peripheral spasm and heightened tension

Overfilling of the bloodvessels from excessive eating and drinking is thought by some to cause arterial sclerosis through constant overdistention of the vessels. In overwork of the vessels and excessive strain

there is heightened tension or increased peripheral resistance, producing the same effect upon the bloodvessels. The result of the above causes is thickening of the intima of the bloodvessels following upon changes in the media and adventitia, and endarteritis deformans occurs in the large arteries.

Symptoms. The symptoms vary. They may be general or local. The disease may be present and the patients die from other causes, and yet the general arterial system is found to be the seat of extensive disease. The local symptoms are due to the local giving way of the vessels in one part, as occurs in apoplexy from cerebral hemorrhage, or the blocking of the coronary artery, or the rupture of an aneurism.

Physical Signs. Arterio-sclerosis is recognized by inspection, palpation and auscultation of the bloodvessels, and by observation of the condition of the heart. The bloodvessels that are visible are elongated and tortuous. There is visible pulsation. When they are palpated the artery is hard; it cannot be compressed; it is corded or rounded underneath the finger, and readily rolled about. The pulse shows at once high tension; the wave is slow in ascent, continues long underneath the finger and subsides slowly. If, in the intervals of the beats the vessel remains full, the pulse, as previously noted, cannot be obliterated. Sphygmographic tracings are characteristic. (See Pulse.) If, after pressure on the radial artery the artery beyond can be felt, its walls are sclerosed; whereas if the artery is obliterated beyond the point of compression, the hardness and firmness of the pulse are due to vascular tension and not to thickened walls. The two conditions should be determined. Hypertrophy of the heart occurs early in the course of the sclerosis on account of peripheral resistance. The hypertrophy involves the left ventricle, and is not attended by dilatation. The apex beat is out beyond the mid-clavicular line; the impulse is heaving and forcible Very characteristic is the occurrence of the second sound at the aortic cartilage. It is clear and ringing; it is heard in the course of the bloodvessels, and is most distinct at or beyond the apex near the heart. Right-sided hypertrophy and dilatation are not generally present. Auscultation of the larger arteries, as the carotids, the abdominal aorta, and femorals, shows a systolic murmur usually rough and high in pitch. All of the above-mentioned conditions may be present, and yet the patient remain in good health. The hypertrophy apparently compensates for the arterial occlusion. There may be no renal disease, or moderate renal cirrhosis may be present, indicated by a transient albuminuria, polyuria, and hyaline tube casts. The subsequent symptoms are due largely to closure of one or more vessels in the peripheral circulation, to the development of an aneurism or dilatation of the aorta, to failure of the hypertrophy of the heart, or to the development of renal cirrhosis.

The blocking of peripheral arteries is due to embolism or thrombosis, more frequently the latter, and to rupture of peripheral vessels, or in all probability, miliary aneurisms. When occlusion of the vessels takes place in arteries which supply the extremities, gangrene may occur. Sometimes the occlusion is due to simple narrowing of the vessels alone. Gangrene of the feet is frequently seen secondary to bad arteries. If the

occlusion takes place in the vessels of the brain, various secondary lesions are produced. In more or less general occlusion from sclerosis of the smaller arteries acute and chronic softening occur. Hemiplegia, monoplegia, or aphasia may occur temporarily from the same cause, relieved by collateral circulation, or permanently from embolism, thrombosis, or rupture of the vessels. Rupture of the vessels, hence apoplexy, is always due to primary disease of the arteries with, in the large majority of cases, miliary aneurisms. If the coronary arteries are blocked, thrombosis with sudden death takes place, or chronic myocarditis, with subsequent aneurism and rupture, occurs. Angina pectoris, with or without thrombosis of the coronary artery, is always associated with arterial sclerosis.

Failure of the hypertrophied heart leads to dilatation with all the symptoms as previously described, including dyspnœa, scanty urine, and dropsies. The murmur at the apex, due to incompetency from dilatation, may simulate chronic valvular disease, which, however, may never have been present. The sclerosis may advance more rapidly in the kidneys than in the other portions of the circulation; on account of the contracted kidney, renal symptoms arise.

Aneurism.

A true aneurism is formed of one or more of the arterial coats. It is usually fusiform, but may be cylindrical. It may be circumscribed or sacculated. The fusiform and the saccular are the forms most commonly seen. False aneurism or dissecting aneurism arises from laceration of the internal coat of the artery. The blood dissects between the layers. It occurs in the aorta. *Arterio-venous aneurism* is seen when communication between an artery and a vein has been set up. If the sac intervenes it is called a *varicose aneurism*. Sometimes a communication is direct, the vein becoming dilated, tortuous, and pulsating. It is known as an *aneurismal varix*.

An aneurism may occur in the course of arterial sclerosis from diffuse distention of the coats. Its typical form is seen in dilatation of the aorta with one or more sacculated aneurisms on its surface. *Sacculated aneurism* occurs from rupture of the tunica media, independent of general disease of the arteries, and in arterial sclerosis. The most common seat is the ascending portion of the aorta It occurs early in the course of arterial sclerosis. Such form of aneurism is seen in the smaller vessels. Aneurisms also arise after the lodgment of an embolus permanently plugging the vessel. The proximal end of the vessel becomes dilated.

Mycotic aneurism, first described by Osler and exhaustively by Eppinger, occurs in malignant endocarditis. The aneurisms are small in size and multiple, not generally recognized during life. They arise on account of the injury produced by the local infection of bacteria in different portions of the vascular system.

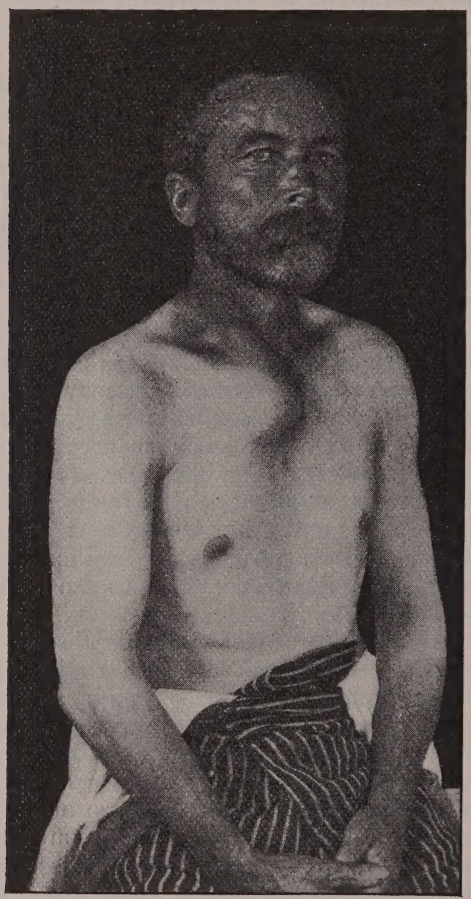
Aneurism of the Aorta. In the thoracic portion of the aorta the causes which produce arterial sclerosis are operative—chiefly physical overwork, alcohol, syphilis, and gout. In this portion of the aorta it may be situated just beyond the aortic ring, at the junction of the

ascending and transverse aorta, in the transverse, or at the beginning of the descending portion. The larger aneurisms are at the two bends of the aorta.

Symptoms. The symptoms of aneurism are largely due to pressure and depend upon the position of the aneurism and the direction of its growth.

Aneurisms, however, may exist without symptoms or appreciable physical signs. Sudden death from rupture may take place in a patient

Fig. 70.



Aneurism of ascending portion of arch of aorta. Tumor in first and second interspaces, extending into neck. Portion of sternum atrophied.

who had been under careful observation on account of concealed aneurism, the presence of which had not been suspected during life. On the other hand, cases occur with characteristic pressure symptoms and with no physical signs. Pressure symptoms depend entirely upon the position of the tumor. Aneurisms of the *ascending* portion of the arch cause dislocation of the heart outward, toward the right pleura or for-

ward, appearing at the second or third interspace, causing erosion of the ribs and sternum. The vena cava is compressed, causing enlargement of the veins of the head and arms; the subclavian vein may be compressed alone, causing enlargement and œdema of the right arm. Localized œdema may result, confined to the thorax (see Œdema). If the aneurism is large the inferior vena cava may be pressed upon, causing œdema of the feet. The right laryngeal nerve may be involved, causing aphonia and dyspnœa. Pain attends the aneurismal process. Aneurisms of the *transverse* portion of the aorta project below, forward, or back. When forward, they produce tumors behind the manubrium which from pressure cause destruction of bone; growing backward, marked pressure symptoms are produced. When the trachea is pressed upon it causes dyspnœa and cough, which is paroxysmal (see Dyspnœa). The œsophagus may be pressed upon, causing dysphagia. The left recurrent laryngeal nerve may be pressed upon, causing paralysis of the corresponding cord, with aphonia (see Larynx). Pressure on the bronchus may produce bronchorrhœa and dilatation, which in turn, may lead to localized abscess. The growth may extend upward, involving the coats of the innominate and carotid arteries on the right side, or carotid and subclavian on the left, markedly interfering with the pulse of the two sides. Pressure on the sympathetic nerve is likely to take place in this situation, with contraction of one of the pupils, although at first it is sometimes dilated. The thoracic duct is sometimes compressed, leading to rapid wasting. In the *descending portion* the pressure signs of aneurism are not so marked. The vertebræ are likely to be pressed upon in this situation. The pain, therefore, is most intense. The œsophagus and left bronchus are compressed. Dysphagia and bronchiectasis, the latter causing bronchorrhœa with subsequent gangrene attended by fever, are liable to occur. The *cough* in bronchorrhœa and the fever, together with emaciation, simulate phthisis, for which aneurism is often mistaken. The physical signs of phthisis are usually pronounced in this situation, and with the presence of bacilli in the sputum, render the diagnosis easy. Rupture takes place into the bronchus or into the œsophagus. In one of my cases, which had been treated for tuberculosis because of small hemorrhages, with the conditions above mentioned, death took place from rupture into the bronchus, causing sudden profuse hemorrhage. When the aneurism is adherent to the œsophagus and slowly ulcerating into it, rupture may take place, followed by instantaneous death. The vertebræ may be eroded and symptoms of spinal compression arise.

I once saw an autopsy performed by a medico-legal expert on a case of sudden death from hemorrhage. The source of the hemorrhage could not be ascertained. There was blood in the stomach. When about to give up the search, the œsophagus and aorta were suggested for examination. A small aneurism was found which had ulcerated into the gullet, with subsequent rupture. In another the aneurism had ruptured into the pleural sac, causing internal concealed hemorrhage with death.

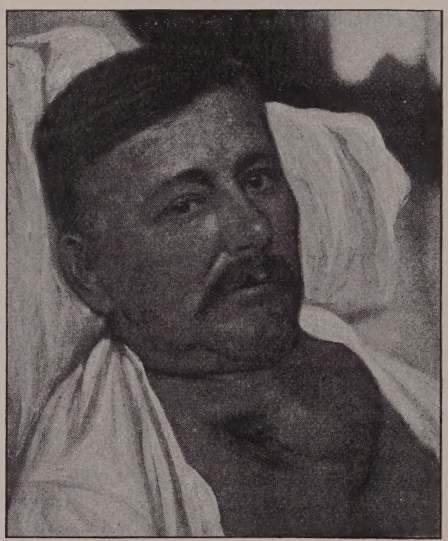
Special Symptoms. While pressure symptoms are the most striking symptoms of this affection, *pain*, which is usually due to pressure, must be referred to. It is an important constant symptom. It is sharp and

lancinating, and may occur in paroxysms. It is more severe and constant when bone is eroded by pressure on the vertebræ or the thorax in front. Anginal attacks may attend the neuralgic pains just described. Pain sometimes follows the course of the nerves, extending down the arm or to the neck or along the course of the intercostal nerves.

If a bone, as the sternum, is perforated, the gnawing pain that attends the ulcerative process is relieved.

Cough. The cough is peculiar. It is paroxysmal and of a brazen, ringing character, in many cases indicating its laryngeal origin, due to pressure upon the appropriate nerves. It is frequently paroxysmal when the pressure is directed upon the windpipe or bronchus.

FIG. 71.



Aneurism of ascending and transverse portions of aorta projecting forward, destroying ribs and sternum. The skin ulcerated, and gradual external leakage took place. The bleeding continued in small amounts for a long time.

In the former instance the cough is dry, in the latter tracheal and bronchial. It is attended by a thin, watery expectoration which, if bronchiectasis with fermentation ensues, becomes thick and ropy. *Dyspnœa* occurs more frequently in aneurism of the transverse portion due (1) to pressure on the recurrent laryngeal nerves ; (2) to compression of the trachea ; (3) to compression of the left bronchus. Marked stridor attends the first form. When one of the recurrent laryngeal nerves, more particularly the left, is pressed upon there is spasm or paralysis of the muscles of the vocal cord, causing hoarseness and loss of voice. Laryngoscopic examination should not be neglected, for paralysis of the abductor muscles without symptoms may be present.

Hemorrhage. The hemorrhage may be gradual when there is small leakage into the trachea at the point of compression. The amount of blood lost is small. It may take place externally (see Fig. 71). Pro-

fuse hemorrhages producing sudden death occur in rupture into the trachea or bronchus and from perforation into the lung. With regard to difficulty of deglutition, it may be said that the sound should never be passed in suspected cases of aneurism on account of the danger of rupture of the sac.

Clubbed Fingers. In intra-thoracic aneurism clubbing of the fingers and incurvation of the nails of one hand are sometimes seen, although comparatively rarely.

Compression and pressure on the *sympathetic system of nerves* has been referred to. In addition to pupillary changes there may be pallor of one side of the face. When the pupil is dilated this pallor may accompany it on account of stimulation of the vaso-dilator fibres. When the cilio-spinal branches of the sympathetic are pressed upon, the dilator fibres are paralyzed. If the pupil contracts there is also hyperæmia of the side of the face and unilateral sweating.

Physical Signs. *Inspection.* In health the position of the aorta cannot be recognized during life. Pulsation may be seen at the episternal notch in rare instances independently of disease of the aorta, particularly in women; it is due to nervous palpitation. An aneurism may exist without any external visible signs. Pulsation may be seen at either side of the sternum above the level of the third rib, most commonly in the second interspace on the right side. The impulse may be seen alone without visible swelling; the chest must be viewed from different situations in order to detect it. An oblique light falling on the surface is sometimes necessary. When the innominate artery is involved the pulsation is observed in the neck, above the sterno-clavicular junction, or above the sternum.

With the abnormal impulse, a swelling or *tumor* is present. It may be large enough to press the upper portion of the sternum and adjacent ribs forward. In other instances a tumor the size of the half of a lemon may be seen along the edge of the sternum. The most frequent site is the first and second right or the second left interspace. The skin over the tumor, as in the case of which an illustration is given, may ulcerate and be the seat of persistent small hemorrhages. The *apex beat* of the heart is displaced downward and outward from pressure.

If the aneurism is seated in the ascending portion of the aorta just beyond the aortic ring a pulsating tumor may be seen in the third interspace at the left edge of the sternum. If in the ascending portion, beyond the heart, the tumor is in the first or second interspace along the right edge of the sternum. In the transverse portion of the aorta the upper portion of the sternum is made to protrude frequently, or the tumor projects upward into the fossæ of the neck. In the descending portion it is in the second or third interspace on the left side. In this portion of the aorta a tumor is seen in the left scapular region in rare instances.

Palpation. Palpation must be employed by the usual method; bimanual palpation must also be used, one hand placed upon the sternum and the other upon the vertebræ. Moderate pressure should be employed. Palpation should also be employed at different periods of respiration. At times signs are only yielded at the end of complete

expiration. It must further be said that palpation must be employed with the tips of the fingers and also with the palm of the hand applied flatly to the surface.

By *palpation* the area and degree of pulsation are determined. If the aneurism is large or has perforated, the impulse is expansile and heaving in character. The sac may be soft and fluctuating, but usually presents considerable resistance. In addition to the systolic impulse the diastolic shock is often felt. This is the most conclusive physical sign. A thrill is frequently present, systolic in time, usually due to dilatation of the arch; at times, to sacculated aneurism. Without visible tumor, pulsation and thrill may be felt in the suprasternal notch, if the head is bent forward so the tissues are relaxed and the finger pushed down toward the aorta. When the aneurism is filled or filling with clot, the tumor may be seen and felt, but be without any impulse transmitted to the hand or thrill felt by the fingers.

Percussion. Percussion forms the most reliable evidence of the presence of an aneurism or aneurismal dilatation in cases in which the tumor is not too deep-seated or small in size. (See Cardiac Percussion.) The area of dulness is increased somewhere in the course of the aorta. It may be observed projecting outward at the right edge of the sternum when the ascending portion of the aorta is the seat of disease, or over the entire upper part of the sternum, extending toward the left, when the transverse portion is diseased. It may be observed as an extension of cardiac dulness upward in the second and third interspaces. Sometimes dulness is detected in the scapular regions, particularly of the left side. The percussion tone is flat, and there is marked sense of resistance. Percussion must be employed with the patient in the upright and in the recumbent posture. The character of the tone and the shape of the dulness must be noted at the end of full inspiration and of full expiration.

Auscultatory *percussion* is of the utmost value, and the method of percussion taught by Sansom and Ewart must be carefully followed. An aneurismal tumor may be present without thrill or murmur, but yield signs of dulness on percussion.

Auscultation. As just stated, murmurs may not always be present. They depend upon the amount of fibrin in the sack. When present the murmur is systolic in time, heard with maximum intensity usually over the abnormal area of impulse or tumor, or over the increasing area of dulness. It is transmitted in the direction of the vessels and may be heard loud in the vessels of the neck and along the course of the aorta. Often a double murmur is heard, the diastolic sound being due to associated regurgitation at the aortic orifice. The diastolic murmur alone may sometimes be heard. Increase in intensity or accentuation of the aortic second sound is pronounced. The sound is ringing in character and is rarely missed in large aneurisms.

The Peripheral Vessels in Aneurism. The pulse in the two radial arteries may show a marked difference both in volume and in time. The difference may indicate the position of the aneurism. If the pulse of the right radial is smaller than the left the aneurism may be in or near the innominate artery; if the opposite, it is near or includes the

orifice of the left subclavian. The difference in time may also aid in the same way to distinguish the seat. Osler refers to obliteration of the pulse in the abdominal aorta and its branches. In one case he could not feel throbbing in the aorta and the femorals, although the circulation was unimpaired. The aneurism was in the descending portion of the aorta, and its pulsation was seen in the left scapular region. The sac was sufficiently large to act as a reservoir which filled during the ventricular systole, and from which the blood poured toward the periphery in a continuous stream instead of being intermittent.

Tracheal Tugging. Tracheal tugging may be obtained in one of two ways. By the old method the patient should be sitting or standing, while the observer sits or stands to one side, and faces him. With the hand furthest from the patient steadying the head, the observer gently but firmly grasps the outer and under surface of the cricoid cartilage with the thumb and finger of the other hand, while the head is slightly thrown back. The head is then flexed so that the neck is no longer stretched. The patient is then told to hold his breath completely, and any up-and-down movement of the trachea is immediately transmitted to the observer's fingers. One must not mistake the transmitted pulsation in the cervical vessels for such movement; and great care should be exercised in seeing that the breathing is entirely stopped.

In the other, or new method, as proposed and practised by Ewart (*British Medical Journal*, March 19, 1892), the observer stands behind the patient, steadying the latter's head against his body, and the cricoid is firmly held between the tips of the first or middle fingers. The writer, after considerable experience, prefers this second method, on account of delicacy of touch, firmness of grasp, and comfort to the patient.

Diagnosis of Aneurism. The special points for diagnosis are: the etiological factors; the antecedent pathological conditions, as arterial sclerosis; the occurrence of pain; the occurrence of pressure symptoms; and the physical signs. These have been sufficiently dwelt upon previously, and it is not necessary to consider them again. It must not be forgotten that aneurism may be present without diagnostic signs, while on the other hand the pressure symptoms may be in abeyance. If one of the two is present in the male subject past forty, with a previous history of syphilis, gout, alcoholism, or muscular strain, the probability is that an aneurism is present. The pressure symptoms always point to some form of intra-thoracic disease which may cause this group of symptoms. Thus, in cancerous disease of the lymphatic glands, or other tumors within the mediastinum, pressure symptoms exactly simulating aneurism may be present and also the physical signs of a tumor. The tumor, however, rarely projects externally, and still more rarely pulsates. If pulsation is present it is not of the expansile character seen in aneurism, nor is there as decided a systolic shock when the ear is placed on the chest. By the same method, shock of the heart sounds is observed. These are notably lessened or absent in tumors from other causes than aneurism. In deep-seated tumors with pressure symptoms the condition of the arteries apart from aneurism is of diagnostic importance. If there is accentuation of the aortic second sound with hypertrophy of the heart,

it points to aneurism. The occurrence of tracheal tugging is a valuable diagnostic point in favor of the latter. In tumor, and especially cancer, there is emaciation and development of a cachexia, which is, as is well known, most pronounced in cancer of the œsophagus. Cancer of the œsophagus from its frequent point of election near the left bronchus often simulates the pressure symptoms of aneurism.

Aneurism must be distinguished from the pulsation of the aorta which is seen in aortic regurgitation. This pulsation is usually associated with dilatation, the latter causing increased dulness, which may add further to the confusion. Exaggerated pulsation without dilatation may, as Bramwell has recorded, be the cause of dulness and pulsation over the aorta. The subjects are under forty, neurotic, and usually anæmic.

In the distinction between pulsating empyema and aneurism usually there is not much difficulty. Wilson points out that aneurism bears a definite relation to the central long axis of the chest. The area of dulness of the aneurism is circumscribed, and is usually the seat of murmurs or other sounds synchronous with the rhythm of the heart. The signs of pulsating empyema are usually upon the left side and at a distance from the median line. The percussion dulness is at the base of the chest and extended. Arterial murmurs are not present. The pulsation is influenced by pressure and by respiratory movements.

In *mediastinal cancer* we are aided by the discovery of enlargement of the glands in the axillary or some other situation, or by a history of the growth elsewhere.

Aneurism must not be confounded with phthisis. The diseased vessel may occlude a bronchus, cause collapse and bronchial dilatation, hemorrhage may occur, bronchorrhœa and cough always ensue Fever is not marked, which fact, with tracheal tugging, vascular physical signs, and the absence of tubercle bacilli, are favorable to aneurism.

Diseases of the Mediastinum.

Inflammation of the mediastinum may be limited to the glands or the connective tissue. Moderate inflammation of the glands, lymphadenitis, occurs in bronchitis and pneumonia, particularly if bronchitis is of specific origin, as in measles or influenza. It is said that such inflammation is of common occurrence in whooping-cough, and may be the exciting cause of the paroxysms. DeMussy and Guitéras have found physical signs of enlargement characterized by dulness in the upper part of the interscapular region in cases of this disease and of influenza. Other authorities, as Osler, dispute the possibility of this occurrence, or at least of its recognition by physical signs. Tuberculous inflammation of the lymphatic glands of the mediastinum may give rise, however, to local physical signs. Abscess of the glands cannot be distinguished during life.

Tumors of the Mediastinum. Cancer and sarcoma are the most frequent forms of tumor of this locality. Hare found the proportion in 520 cases to be as follows: 134 of cancer, 98 of sarcoma, 21 of lymphoma, 7 of fibroma, 11 of dermoid cyst, 8 of hydatid cyst, and the remainder

of lipoma, gumma, and enchondroma. The tumor is most frequently found in the anterior mediastinum when one region alone is affected. The disease may be either primary or secondary. In sarcoma the former variety is more frequent. Males are chiefly affected, and the age of onset is between thirty and forty.

The symptoms of mediastinal tumor are chiefly due to pressure. Dyspnœa is early and constant, and may be laryngeal, or from pressure on the trachea. In some instances, encroachment upon the heart or the vessels causes dyspnœa. Again, it may be due to a pleural effusion which accompanies the growths. Cough of a peculiar character occurs. It is laryngeal, and of a dry, brazen quality. For the same reason there may be aphonia. (See Disease of the Larynx.) If the blood-vessels are pressed upon, symptoms of obstruction occur dependent upon the vessel occluded. Œdema of the upper extremities may occur. If the œsophagus is pressed upon there is difficulty in deglutition. In some instances the sympathetic nerve is pressed upon, causing hyperæmias and pupillary changes.

CHAPTER IV.

DISEASES OF THE MOUTH, FAUCES, PHARYNX, AND ŒSOPHAGUS.

The Mouth.

THE mouth is affected by comparatively few diseases, most of which are dependent upon the influence of micro-organisms. The cavity forms a good breeding-place for all forms of organisms, and were it not for the secretions and constant cleansing of the mouth by the passage of food and its physiological labors, diseases would be very common. Indeed, it is possible that such diseases do not take place at all unless there is some perversion of the normal secretion which destroys its antiseptic or anti-microbic power. We know but little specifically concerning the changes in the secretions. Clinically we do know, however, that in conditions of poor nutrition, in wasting diseases generally, and probably in connection with the rheumatic diathesis, there is such change in the secretions as permits pathogenic micro-organisms to exercise their influence upon the mucous membrane. The result of their action is seen in various forms of inflammation.

Symptomatology. The symptomatology of mouth affections is the symptomatology of inflammation. Pain, heat, redness, and swelling abound.

THE SUBJECTIVE SYMPTOMS.

The subjective symptoms are not characterized by great gravity, but they are most annoying.

PAIN. This symptom is most aggravating because it is excited by the many functional acts connected with the mouth. It occurs in all inflammations and ulcerations except those due to syphilis. It is aggravated by food, by movements of the lips, cheeks, or tongue, and by attempts to discharge saliva. The absence of pain is observed in gangrene.

HEAT. The patient complains of the heat of the mouth in inflammations.

DRYNESS. This symptom is complained of in fevers and in those who are compelled to sleep with the mouth open. It may be a condition of itself, as the following shows.

Dry Mouth. Hutchinson first described a condition of the mouth in which dryness was the chief complaint. The secretions are suppressed entirely, the tongue red and dry, the mucous membrane of the cheeks and palate smooth, shining and dry. Functional movements are very difficult. The majority of the cases are in women in whom

the general health is always impaired. It is thought to be of central origin by Hayden, on account of which the secretion of the salivary and buccal glands is modified. A moderate amount of dry mouth occurs in fevers. It is also symptomatic of chronic gastritis, and may occur in diabetes.

The Objective Symptoms.

The objective symptoms are determined by inspection and palpation. By such research the color of the parts of the mouth is observed, and changes in temperature, and in the size and shape (swelling) are ascertained. The teeth, gums and tongue are also examined.

Color. The normal redness of the mucous membrane may be increased or lessened in hue. Pallor is associated with anæmia. Increased redness attends inflammation, and with it the temperature is raised. The mucous membrane is yellow in jaundice, bluish in cyanosis. Both of the latter changes may be observed to greater advantage under the tongue. The mucous membrane is the seat of pigmentation in Addison's disease and in argyria. In the former, small oval purplish spots are seen. They must not be confounded with the pigmented spots common after stomatitis in negroes. Eruptions occur in the mouth and may precede external eruptions. This is notably so in measles. In this affection the eruption is seen on the hard and soft palate twenty-four hours before the development of the rash. In smallpox and chickenpox the vesicles are seen.

Shape. Swellings are seen usually from disease of structures about the mouth. The floor of the mouth is invaded by glands underneath or swelling of the cellular tissue. Bone diseases and some teeth affections cause swellings. The dental arch must be observed. Increase in height of the arch is due to adenoid disease or to the habit of thumb-sucking in childhood, much more likely the former.

Fœtor. The odor imparted to exhaled air is peculiar in mouth-affections. It may be a simple fœtor or of a metallic or gangrenous odor. Fœtor attends all inflammations; it is more pronounced in ulcerative and mercurial stomatitis. In the latter it may be metallic.

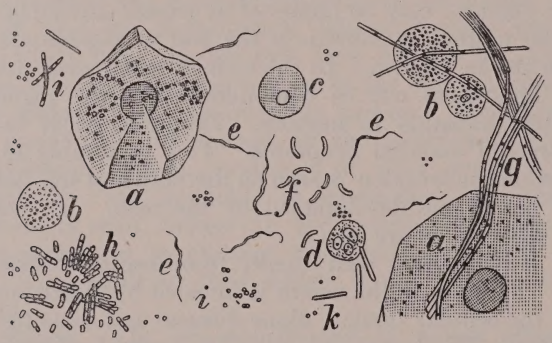
Salivation. Increased flow of saliva occurs in all inflammations unless attended by high fever. It may be constantly discharged by the patient or dribble in a continuous stream. (See Saliva.)

Secretions of the Mouth. The saliva is derived from the parotid, submaxillary and sublingual glands and from the mucous glands within the mouth. The mouth should be washed with warm alkaline solution and afterward with cold water, in order that the saliva obtained may be perfectly pure. After the washing the glands may be stimulated by the application of dilute acid on a glass rod. The normal amount that is secreted in twenty-four hours varies from two to three pints. It is of a light bluish color, or is colorless. It is somewhat stringy. On standing, two layers form in a conical glass, the upper clear, the lower cloudy. The reaction of saliva is alkaline.

Microscopic Examination. The following formed elements are observed: 1. Salivary corpuscles of the appearance of, but larger and more granular than a white corpuscle. 2. Epithelium. The squamous

variety derived from the mouth is seen. The cells are large in size and of polygonal shape. 3. Fungi. In health the mould and yeast fungi are seldom found. In disease they are present in large numbers; fission fungi are met with in great numbers, both in health and in disease. In health small and large colonies of micrococci are found along with abundant bacilli. Miller has studied the micro-organisms of the mouth carefully and exhaustively (see *The Dental Cosmos*), both by microscopical examination and culture methods. The following are found to be pathognomonic: (1) The leptothrix buccalis; (2) vibrio buccalis; (3) spirochæte dentium; (4) micrococcus tetragenes; (5) the micrococcus de la rage; (6) the micrococcus of septicæmic sputa; (7) the bacillus of decaying teeth, three varieties of the staphylococcus; (8) the bacillus crassus sputigenus; (9) the bacillus salivarius septicus and bacillus septicus sputigenus.

FIG. 72.



Buccal secretion. (Eye-piece III., obj. Reichert, 1/15, homogeneous immersion; Abbe's minor, open condensers.) Friedlander's and Gunther's method. (VON JAKSCH.)

a, epithelial cells; *b*, salivary corpuscles; *c*, fat drops; *d*, leucocytes; *e*, spirochæte buccalis; *f*, comma bacilli of mouth; *g*, leptothrix buccalis; *h*, *i*, *k*, different fungi.

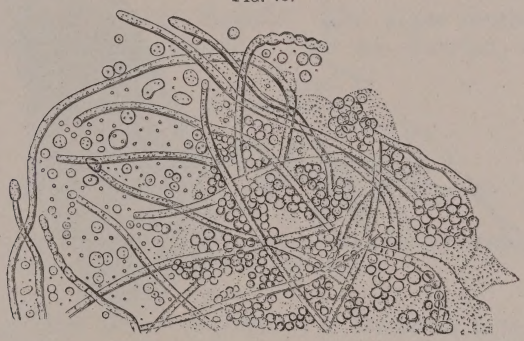
Of course in the saliva the thrush fungus, actinomyces, the tubercle bacillus and the bacillus of diphtheria are found. It must not be forgotten that the diplococcus pneumoniæ or micrococcus lanceolatus, which is the specific cause of pneumonia, is found in the saliva in health. It is also called the bacillus sputi septicæmici.

Chemical Examination. The chemical characters of the secretion depend upon the activity of the different glands. The saliva contains a trace of albumin, found by heating; a ferment which changes starch into sugar; mucin; and sulpho-cyanide of potassium occasionally. In disease, as the quantity is diminished rather than increased, examinations have rarely been made. In *ptyalism* the saliva should be collected after rinsing the mouth frequently, especially after eating. The reaction is found to be alkaline and of low specific gravity, 1002 to 1006. Albumin is tested for by the usual methods. The sulpho-cyanides are detected by a solution of the chloride of iron. When this is added to the fluid a bright red color appears which does not disappear with heat; a similar color may be obtained by the same test from the saliva in opium poisoning, due to the precipitation of meconic acid.

Sugar is tested for by methods used in the examination of the blood for this substance. The diastatic ferment is detected by adding 5 c.cm. of saliva to 50 c.cm. of starch solution, placing the mixture in a warm chamber or a water bath heated to 40° C. After an hour's time the fluid will show the presence of grape sugar. Nitrites are detected by adding a little of the saliva to a mixture of starch paste, iodide of potassium, and dilute sulphuric acid. If the nitrites are present a blue color results.

Saliva in Disease. Increase and diminution in the amount of saliva will be referred to. In catarrhal stomatitis the secretion is increased. It is acid and contains epithelium in excess. In ulcerative stomatitis it is also increased, is of a dark brown color, fœtid and alkaline. It contains degenerated epithelium, leucocytes, blood corpuscles and many forms of fungi. It is increased in pregnancy, in rabies and glosso-labio-laryngeal palsy. I have seen it in excess in the convalesence of typhoid fever. It is increased by the internal use of jaborandi.

FIG. 73.



Oïdium albicans, the vegetable parasite of muguet or thrush. (Reduced from CH. ROBIN.)

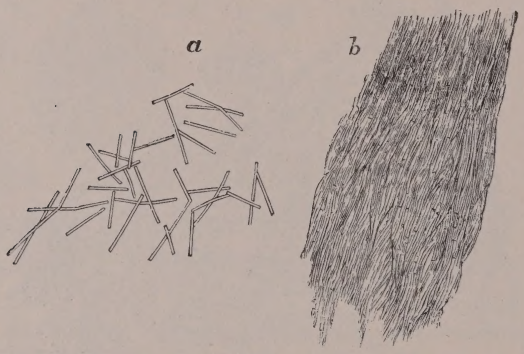
The reaction becomes acid in diabetes, gout, rheumatism, and mercurial poisoning. Urea may be found in cases of nephritis, particularly in uræmia. There is no sugar in diabetes. Fenwick has investigated the changes in the sulpho-cyanide of potassium in disease. By a scale of colors he was enabled to compare the saliva in which sulpho-cyanide of potassium had been detected in health with the saliva in various diseases. He believes that the amount of this ingredient is indicative of the degree of functional activity of the organs of nutrition. It is increased in acute inflammation and in the earlier stages of cancer and phthisis; in acute congestion of the liver from stimulants or food excess; and in rheumatism, gout, and the convalesence of typhoid fever. Where the power of the nutritive organs is diminished the sulpho-cyanide is lessened, as in late phthisis and cancer, the later stages of chronic diarrhœa and dysentery, chronic catarrhal jaundice, in ascites, and in the passive congestion of the abdominal viscera. Fenwick believes tedious recovery and frequent relapses will occur if this element is found in excess in acute rheumatism.

Thrush. The fungus peculiar to this disease is found. Saliva is increased; it is usually acid. The disease is characterized by the for-

mation of small patches on the mucous membrane, which in a few days coalesce and form a mass which may cover the entire mouth and extend to the fauces. Before coalescing they are firmly adherent. Subsequently they loosen. On microscopical examination, in addition to epithelial cells, leucocytes, and unorganized elements, the characteristic parasite is seen. It is in ribbon shape, composed of long segments containing often highly refractive nuclei at either end of the segment. The parasite varies in length, but is made up of many segments which are shorter toward the extremity. The segments vary in length. They are homogeneous. When mounted in glycerin they are readily seen. Spores are also seen.

The Leptothrix Buccalis. The latter is seen in ribbon-like bundles composed of various segments. They stain bluish-red in potassic iodide solution. It is most frequently seen in the tartar of the teeth.

FIG. 74.



Leptothrix buccalis from the gums at edges of teeth. *a*, the filaments separated; *b*, masses of filaments. × 350.

THE GUMS. The color and consistence is inquired into. The former changes with changes in the mucous membrane of the mouth, in inflammations and ulcerations, and in certain metallic poisonings. The gums swell and grow spongy in inflammations.

The Gingival Line. In cases of tuberculosis a red line at the junction of the gums and the teeth is frequently seen. At one time it was thought to be of diagnostic value. It is seen, however, in other cachectic conditions, as carcinoma, and at times in diabetes.

The Gums in Scurvy. In scurvy the gums are swollen and spongy. They bleed easily, and usually are streaked with blood. Ulcers form along the teeth line. There is not much fœtor of the breath. In mild cases the inflammation may be limited to the gums of four or five teeth only. The gums about teeth that are decayed are usually the seat of the most marked inflammation.

The Gums in Lead-poisoning. In chronic lead-poisoning a blue line appears on the gums and margins of the teeth. The line is preceded by a row of separate black dots occupying the seat of the papillæ of the

mucous membrane. It does not always extend along the entire margin, but may be limited to a few front teeth in either the upper or lower jaw. In the more advanced cases there is some salivation and sweetish metallic taste in the mouth and metallic fœtor of the breath.

THE TEETH. In all diseases of the gastro-intestinal tract it is important to investigate the state of the teeth. Cases of indigestion are often due to defective mastication rendered so by decayed teeth. Caries of the teeth may cause headaches or neuralgias, near or remote (see Headache), and may explain many cases of foul breath. Pitting of the surface of the teeth, and thinning of the enamel in transverse grooves, are held by some to be due to mercury. It is no doubt true that infantile stomatitis independent of mercury is the cause of these changes. They must be distinguished from the so-called Hutchinson's teeth. In stomatitis which affects the teeth the molars are honeycombed to the greatest degree, the incisors affected next. In addition to pitting and erosion the color may be darker. A transverse furrow crosses all the teeth at the same level.

The Teeth of Congenital Syphilis. The upper central incisors of the permanent set are affected. They are dwarfed, narrowed and

FIG. 75.

Notched teeth. Malformation of permanent teeth found in hereditary syphilis. (MR. JONATHAN HUTCHINSON.)

short. The middle lobe of the tooth is so atrophied as to leave a single broad vertical notch in the edge of the tooth. A narrow furrow passes upward sometimes from the notch on both anterior and posterior surfaces nearly to the gum.

It is seen from the above that the appearances of the first set of teeth may be an index of the condition of the nutrition of the child in infancy.

Teething. During the period of infancy it is well to remember the influence of the eruption of the teeth upon the general constitution. While many prominent authorities believe that the eruption takes place without the occurrence of general or reflex symptoms, on the other hand equally careful observers believe that nervous phenomena often attend the process. The latter class of observers attribute the feverishness, restlessness, loss of appetite, and gastro-intestinal disturbance to this cause. Convulsions at this period are believed to be due to the pressure of the tooth, which cannot break through the mucous membrane, upon highly sensitive nerves at the root. Even in later life reflex convulsions are held by some to be due to teeth.

Slowness in the development of the teeth may be due to rhachitis, which should be looked for. The student should be familiar with the periods of development, the number of teeth that appear at each period, and the date of the eruption.

DATES OF ERUPTION OF THE TEETH.

Milk Teeth.

$$\frac{2M\ 1C\ 4I\ 1C\ 2M}{2M\ 1C\ 4I\ 1C\ 2M} = 20$$

Eruption of	central incisors	about . . . . . 7th month.[1]
"	lateral incisors	" . . . . . 9th month.
"	first molars	" . . . . . 15th month.
"	canines	" . . . . . 18th month.
"	second molars	" . . . . . 24th month.

Permanent Teeth.

$$\frac{3M\ 2B\ 1C\ 4I\ 1C\ 2B\ 3M}{3M\ 2B\ 1C\ 4I\ 1C\ 2B\ 3M} = 32$$

Eruption of	anterior molars	about . . . . . 7th year.
"	central incisors	" . . . . . 8th year.
"	lateral incisors	" . . . . . 9th year.
"	anterior bicuspids	" . . . . . 10th year.
"	posterior bicuspids	" . . . . . 11th year.
"	canines	" . . . . . 11th year.
"	second molars	" . . . . . 12th to 14th year.
"	third molars (wisdom teeth)	about . . . . . 18th to 25th year.

Stomatitis.

This inflammation is not limited alone to the mouth, but extends to structures within the mouth, as the gums, and may invade the tongue. The inflammation is recognized by the subjective and objective signs common to such inflammations. There is pain, and hence the child (for it usually occurs in children) refuses to nurse or take the bottle, or cries when food is given. The pain is accompanied by fœtor of the breath. This occurs in all forms of stomatitis. Its origin, as well as the origin of the pain, is readily determined by inspection.

On inspection we note the usual signs of inflammation. It is rare for the latter to be general; i is localized to small areas which rapidly become ulcerated. When general the mucous membrane is red and hot; the color extends to the sides of the gums and lips and tongue. This is seen in the *catarrhal* form, and in addition the follicles are enlarged. The tongue becomes red and smooth, or may be covered with a white coating through which the prominent red fungiform papillæ project. Attendant on inflammation there is increased secretion, which dribbles from the mouth, or is constantly discharged by older patients. The red hue of the mucous membrane is attended by swelling. The heat of the mouth is sufficient often to raise the temperature of the expired air so that the breath is hot.

APHTHOUS STOMATITIS. When the inflammation is more intense in local areas ulceration takes place. Thus in *aphthous stomatitis* small yellowish-white spots appear, at first discrete, but soon dotted over the mucous membrane inside of the cheeks, in the roof of the mouth, along the sides of the gums and on the tongue. They subsequently break down into shallow ulcers with raised red margins.

ULCERATIVE STOMATITIS. The disease occurs in ill-nourished subjects and is often intercurrent with exhaustive diseases, as chronic

[1] Lower incisors first.

diarrhœa. It may be seen in epidemic forms in camps, and in penal and other institutions on account of unsanitary conditions. In *ulcerative stomatitis* the inflammation is more pronounced on the gums. They are swollen, red, and covered with ulcers. The gums which are filled with teeth are affected, and the ulcers are usually at the gingival border. The ulcers are covered with yellowish material. The flow of saliva is much increased in this affection. It is acid in reaction. The submaxillary glands are enlarged. The fœtor of the breath is very great.

Thrush. In *parasitic stomatitis*, or *thrush*, raised white patches are seen looking like small curds of milk. The patches vary in size, and upon the tongue may cover an area as large as a three-cent piece. The white patches are distinguished from milk curds because they cannot be removed by the napkin or brush. It has been thought that the parasite which is the cause of the inflammation is the *oïdium albicans*. But Forchheimer prefers to group it under saccharomyces.

Stomatitis Materna. Painful ulcers occur in the mucous membrane of the lips and cheeks in nursing women. They are solitary and interfere with mastication.

Gangrenous Stomatitis. The affection appears as a gangrenous inflammation of the gums, mucous membrane, and deeper tissues of the cheek. At first a small, dark red, hard spot is seen, which increases in size, and becomes of a purplish color. The cheek rapidly becomes swollen, tense and brawny. On the surface of the more indurated portions a bleb forms which soon breaks with rapid ulceration. The ulcer is dark and gangrenous and soon perforates the cheek. It extends to the jaw, followed by necrosis of that bone. The characteristic odor of gangrene attends the process. While the affections previously mentioned are generally dependent upon poor nutrition, *gangrenous stomatitis* is always secondary to depraved, depressed, or debilitated states of the system. Several cases may occur at the same time among children congregated in institutions in which there are bad hygienic conditions and to whom improper food has been given.

Mercurial Stomatitis. The gums are involved in ulcerative stomatitis, and mercurial stomatitis, or ptyalism, particularly affects these structures. It also involves the salivary glands. The inflammation is caused by mercury. It may occur from the medical use of the drug, particularly in persons who are unduly susceptible, or are not particular in regard to mouth-cleansing. The inflammation is painful and attended by profuse discharge of saliva, hence the name, *salivation*. The tongue is swollen, marked on the sides by the teeth, and may be protruded with difficulty, on account of its size. It is tender to the touch. It is covered with a heavy, creamy coating. The gums are swollen, red, sore, and bleed on the slightest touch. Ulcers along the border occur, may become extensive, and in some instances extend to the jaw. The teeth become loosened. The fœtor of the breath is heavy, offensive, and of a metallic character. The inflammation is usually preceded by a metallic taste in the mouth, and the patient notices pain on mastication, which increases in severity as the inflammation develops. In mild cases it is limited to the gums, in others the

tongue and salivary glands and the mucous membrane of the mouth are affected.

Ulcers. In addition to the forms just described of ulcerative inflammation of the mouth, *ulcers* are seen, such as herpes secondary to gastric disturbance, and more particularly the ulcers that attend syphilis. In the secondary stage of syphilis, mucous patches are seen as bright red, symmetrical, oval or crescentic patches or erosions. They are generally covered with a scanty grayish-white secretion. They are not generally painful. They occur on the mucous membrane, and at the same time may be found on the tongue and fauces.

Sublingual Ulcer. This local ulcer is on the frænum of the tongue. It is seen in whooping-cough, and is due to the rubbing of the tongue against the teeth in the act of coughing.

The Tongue.

Examination of the tongue is made for diagnostic purposes with a greater show of wisdom on the part of the examiner, and greater satisfaction to the patient, but with less satisfactory results from a diagnostic standpoint, than the examination of any other portion of the body. Examination is resorted to because the mucous membrane of the tongue is the only mucous membrane of the body, except the oral and faucial, which is open to inspection, by which we judge the effects of general diseases upon mucous membranes. Because of its relations with the gastro-intestinal tract it is thought to be indicative of disorders of that tract. Recent studies promulgated by Hutchinson, Butlin, and other observers have resulted in views at variance with the above. Both the distinguished gentlemen above mentioned are surgeons, and look upon the tongue as a local organ. Investigating it as such they concluded that the changes in the coating, which had been considered to have so much clinical significance, depended largely upon parasitic invasion, and were not due to changes in the epithelium. The parasitic invasion, they hold, is largely dependent upon local conditions, which, however, it is true, are on their part dependent upon a state of the system. Since the writings of Hutchinson and Butlin, Dickinson returned to the investigation on the lines laid down by older teachers, and has, in a measure, restored the tongue to its original position as a diagnostic factor in an estimation of the state of the general system and in diseases of the gastro-intestinal tract.

We study the tongue with a view to ascertaining its color; the character of eruptions if present; the occurrence of indentations, excoriations, furrows or fissures; the occurrence of ulcers and of patches. Plaques, nodes and nodules are also seen on the tongue. *Inflammation* of the tongue occurs, and it is the seat of *atrophy* and *hypertrophy* and of various *tumors* in the parasitic diseases. The *movements* of the tongue are also observed, as an indication of the power of muscles which are under centric influence closely related to important centres in the medulla oblongata. Surgical affections of the tongue will not be considered; local affections will only be referred to in connection with general diseases.

Discolorations of the Tongue. Yellowish-white, oblong patches, soft, but slightly raised, are sometimes seen along the sides of the tongue —*xanthelasma*. They are sharply defined and vary in size from a split pea to a three-cent piece. Xanthelasma is also situated upon the eyelids and upon the palms of the hands, and rarely in other portions of the body. It occurs in jaundice or in persons who are said to be subject to bilious attacks.

Addison's Disease. Dark purple or black marks are seen on the tongue as well as on the surface of the lips. They are sharply defined, neither raised nor depressed, and vary in size. They are dark purple in color. Unless associated with discolorations of the skin they are not of significance. They are also seen on the tip of the tongue of a bluish-black color, while the patches inside of the lips and cheeks are brown. *Blood-stains* are observed in purpura. Bright red spots the size of a split pea, or patches, or *ecchymoses*, are of frequent occurrence. The color of ecchymoses is not removed by pressure. Hemorrhagic *infarcts* are sometimes seen on the tip of the tongue.

Black Tongue. This rare condition is of parasitic origin. It has recently been described anew by Cohen. It is also known as *nigrities*. The affected portion is of brownish-black or black color, varying in size and usually seated on the middle of the dorsum of the tongue. It looks like an iron-stain, and in some instances the surface is roughened. The papillæ are abnormally enlarged. It usually begins as a small spot, and extends slowly so that at the end of a month the dorsum is covered. The centre is blacker than the circumference. After the entire dorsum is covered the spot begins to disappear from the circumference toward the centre, and is followed by desquamation. This series of phenomena is repeated and the entire affection subsides slowly. Desquamation may last from a few days to two months. The papillæ of the affected surface, too, look like "a field of corn laid by the wind and rain." The sensations of taste and touch are not altered, but a sensation of dryness is marked. It must be remembered that a black tongue is sometimes the result of deliberate deception.

Inflammation of the Tongue. *Acute glossitis* is a rare affection, more common in adults than in children, and more frequent in men than in women. It occurs more frequently in the summer. The onset is rapid. After a short period of tenderness on mastication, the movements of the tongue are stiff or painful, or there are pains in the muscles of the neck and submaxillary region. In a few hours the tongue swells. It rapidly increases, and at the end of fifteen to twenty hours is three times its natural size, protrudes from the mouth, is indented by the teeth, and is almost immovable, feeling heavy, painful and tender. It is coated with a thick fur on the dorsum. Salivation accompanies these symptoms, speech is impossible, dysphagia extreme and dyspnœa not unusual. The glands underneath the jaw are swollen. The temperature rises to 101°, rarely above it, even if the case is severe. Death may occur in a few hours from suffocation, or after a longer interval, from diffuse suppuration, exhausting septic fever, or pneumonia. Gangrene is more frequent than spontaneous resolution. The swelling begins to subside in three or four days. Small ulcers

form on the surface of the tongue, and by the end of a week its normal aspect is regained. The fever and distressing symptoms subside along with the local swelling. It is said to be due to colds, to bites and stings of animals, to mercury, and to corrosive and acrid substances. It may occur in fevers. The diagnosis is easy. It may be difficult to distinguish it from acute œdematous swelling due to salivary calculus or affections of the floor of the mouth. Acute *ranula* sometimes causes considerable swelling of the tongue, simulating acute glossitis. *Hemiglossitis* sometimes occurs. The local symptoms are not so great, because half of the mouth is occluded only. I saw a case in my early connections with the University Dispensary in which the inflammation was limited to half the side of the tongue on the posterior surface. It went on to suppuration, but was not attended by serious symptoms, except discomfort in eating. It was preceded by a definite nodule in the substance of the inflamed part. Glossitis from mercurial poisoning has been described in connection with stomatitis.

Chronic Superficial Inflammation of the tongue may also occur. The surface is smooth and deprived of papillæ over the affected area, which is redder than natural. The margin of the raw patch is sharply defined, but the area has no depth. The epidermis alone is removed. When associated with dyspepsia it covers a considerable area of the surface of the tongue. It may be deprived of papillæ on the front part of the dorsum while the fungiform papillæ remain. One observer, Hack, has described these ulcers as peculiar to certain females. In three generations of two females he observed a row of long, oval areas. They commenced in early childhood. The tongue was strikingly smooth over large areas, with red excoriations here and there. There was no syphilis. In chronic superficial glossitis, excoriations are due to slight traumatism or to dyspepsia.

Eruptions. Eruptions of variola, measles and erysipelas are seen on the tongue. Herpes and aphthous ulcers, preceded by vesicles, are situated on the surface of the tongue.

Indentations occur when the tongue is swollen, as in mercurial and other forms of glossitis. The borders of the tongue are indented by the pressure of the teeth. In states of debility a flabby tongue with indentation of its borders is often seen. Sometimes the indentations are so pronounced that the pressure of the teeth causes ulceration.

Excoriations on the surface of the tongue, or rawness, arise from injury and may be seen in dyspepsia.

Furrows, or Grooves and Wrinkles, are seen on the dorsal aspect of the tongue. They are not necessarily tokens of disease; in many persons they are of constant occurrence. *Furrows* vary from a few lines to an inch or more in length. In many this is most striking in the middle line of the tongue. The median furrow is liable to become ulcerated on slight provocation. The edges of the fissures are smooth and without papillæ or fur. Other furrows are directed longitudinally and vary in depth. They may be curved and forked. They are more frequent in older persons, especially if the tongue is too large to lie within the circle of the teeth. They are an evidence of past inflammation, or rarely of hypertrophy. They resemble the median

furrow as regards smoothness and absence of fur. Inflammatory furrows occur in chronic superficial inflammation, but more commonly after chronic inflammation which has left the tongue enlarged. The furrows are sometimes so abundant that the surface of the tongue looks like the eyelid. The raised areas become sore, due to irritation of a foreign body (food) or a tooth. They are an indirect result of inflammation. True inflammatory furrows, described as *dissecting glossitis* by Wunderlich, occur. Dissecting glossitis is only a more aggravated form of superficial glossitis. Furrows of this character may be due to syphilis, and dissecting glossitis sometimes has a syphilitic origin. Fissures and clefts are usually caused by the rubbing and deep indentation of a rough and jagged tooth. The dental fissure may be inflamed around it and be seated on an indurated base. The sides and bottom are ulcerated. It is recognized by its association with the offending tooth. It may be mistaken for syphilis, which is a common cause of fissures.

Syphilitic Fissures. In secondary syphilis they are always on the borders of the tongue; they are almost certain to occur if the teeth irritate the border. They may be due to the ulceration of a mucous tubercle which is developed upon the border of the tongue. The ulcer is stellate, and gradually deepens until it becomes a foul fissure. Two processes cause the ulceration—syphilis and the irritation of the teeth. It must be remembered that the tongue is always predisposed to inflame and ulcerate in syphilis. Syphilitic ulcers are not very angry like non-syphilitic sores and fissures which are produced in persons out of health. They may be sensitive, however, on account of the involvement of the tongue. The absence of active inflammation, the large number of the sores and fissures, and association with other signs of the disease upon the tongue, cheeks, and lips point to their syphilitic origin. Tertiary syphilitic ulcers are more pronounced and deeper than other forms. They may be as long as two or three inches; they are sinuous and branched. Gummata may occur on the tongue at the same time. The gummata may be localized or arranged in lines which break down. *Sclerosis* of the tongue, as described by Fournier, follows the healing of these ulcers. It is curious to note that the lymphatic glands are seldom enlarged in association with syphilitic fissures. The fissures must be distinguished from *carcinoma* and *tuberculosis*. In carcinoma there is a distinct tumor, which may become fissured. Tuberculous ulceration is a sign of association of tubercle in other organs. The tuberculous fissures are small, at first single; tubercle, however, rarely begins as fissures, but as tuberculous ulcers on the tip or borders of the tongue. They are stellate or irregularly branched. They are shallow at first, and deepen later, but do not widen in a corresponding manner. The lymphatic glands are always involved (see Tuberculous Ulcer).

Ulcers of the Tongue. They may be simple, aphthous, or traumatic. *Simple ulcers* follow long-standing superficial glossitis. They form in the centre of the tongue, or of the diseased inflammatory area. They are due to sloughing, or simple melting away of epithelium. The ulcer is smooth, red, glazed on the surface. The edges are callous and inactive, the shape is irregular. It is sensitive, and may be pain-

ful. The signs of chronic glossitis continue with it. *Dyspeptic* or *catarrhal* ulcers occur on the tip or on the dorsum near the tip. The dorsum of the tongue from the tip extending back is very red, and filiform papillæ are absent. They are small, superficial ulcers without definite shape or character, except that they are red and irritable. Dyspeptic ulcers may occur from the breaking down of vesicles of the tongue. They are small, circular, well-defined ulcers, with sharp-cut edges, in size from a pin's head to a split pea, and are the source of considerable pain and much annoyance. They are recurrent. Salivation may attend them. *Aphthous ulcers* are seen in children and adults, and are attended with the same symptoms as aphthous ulcers of the mouth, with slight fever. Fœtor is characteristic. *Traumatic ulcers* from sharp teeth may persist a long time if the general health is bad. When active, they may be mistaken for syphilitic sores, and when indolent for syphilitic, tuberculous, or cancerous ulcers. The rapidity of formation, the location opposite a rough tooth, and the absence of other signs of syphilis point to its true nature. *Chancre* must be excluded by the greater hardness and circumscription of the lesion, its seat near the tip, its association with enlargement of the lymphatic glands. The latter is not present in traumatic ulcer, unless it is acute and angry. It is distinguished from tuberculous ulcers by the absence of signs of tubercle in other organs and by the result of an examination of the scrapings of the ulcer; from cancer by the age. In cancer, all the glands become affected later.

Tuberculous Ulcer. The tuberculous ulcer presents an uneven, pale, flabby surface, covered with a yellowish-gray viscid or coagulated mucus. The edges are sometimes sharp-cut, sometimes bevelled, seldom elevated. They are not usually very red. There is but little surrounding inflammation, and the adjacent portions of the tongue are but slightly swollen. The borders of the ulcer may be sinuous, and the shape oval or ovoid, or elongated. In the neighborhood of an ulcer a number of tiny yellowish-gray points may be observed. The ulcer is painful, and attended by salivation. I saw in the Philadelphia Hospital a case of tuberculous ulcer of the tongue, in a young man twenty-five years of age, with pulmonary and intestinal tuberculosis. The dorsum of the tongue was covered with a dozen ulcers with sharp-cut edges and pale, flabby granulations, without induration or inflammation around them. They were yellowish gray, and in scrapings of them, tubercle bacilli were found. Tubercular ulcer must always be diagnosticated from syphilitic and cancerous ulceration. The associate symptoms are often most reliable. Ulcers due to lupus are also seen upon the tongue.

Patches and Plaques. Space forbids further consideration than the naming of the plaques which are seen on the tongue. First, there is the smoker's patch on the middle part of the dorsum about the point where the tobacco-pipe rests, or where the stream of smoke from the pipe or cigar strikes the tongue. This is a slightly raised area of oval shape. It is not ulcerated, but is smooth and red, or livid. Sometimes it is bluish-white or pearly in appearance. The smoothness is characteristic. White and bluish-white patches or plaques are seen in *leucoma*,

leucoplakia, ichthyosis, keratosis, and are also known as opaline plaques. The smoker's patch belongs to the same class, and is probably an early stage of the affections. It means a whiteness, or white opacity of the surface of the tongue, usually on the dorsum. It is almost always the result of the direct action of irritants. These patches are unknown under twenty years of age, do not commence after sixty, and very rarely attack women. They are not attended by subjective symptoms usually. There may be a sensation of induration and dryness. The course is always chronic.

Chronic Superficial Glossitis. The whole dorsal aspect of the tongue is smoother than natural, the mucous membrane is redder than normal, the surface uneven. The papillæ have disappeared. Excoriations and superficial ulcers usually accompany the inflammation. The tongue is enlarged and its borders marked by the teeth. The surface looks glossy. The tongue feels stiff and uncomfortable. Movement is irksome, irritating foods are hurtful. Spirits and tobacco cause distress. Indiscretions in diet quickly produce fresh inflammations.

Wandering Rash. Ringworm, or circular exfoliations occur most frequently in children. One or more patches on the surface of the dorsum of the tongue are observed, smooth and red, but not depressed or elevated. The filiform papillæ have been shed. The patch spreads and becomes a ring, circular or oval. The border is faintly or decidedly yellow, and usually slightly raised and sharply defined. The circles may widen and contract from time to time. No subjective symptoms are noted except itching in a few cases. The cause is not known. The diagnosis is easy. It may continue for months or years.

Mucous Patches are multiple lesions of syphilis in the mucous membrane. They have been referred to in the section on Diseases of the Mouth, and further reference to them will be found in works on surgery.

Nodes or nodules in the tongue are always tuberculous or syphilitic.

Atrophy of the tongue is very unusual; hemiatrophy may occur as the effect of central or peripheral causes, as softening, hemorrhage, or tumors of the region of the hyperglossal nucleus. Other centres near the nucleus are affected, hence other forms of paralysis are seen, due to the lesions of the medulla. These are seen in progressive muscular atrophy and bulbar paralysis, and in cases of hemiplegia. It is not difficult to recognize it on inspection. The functions of the tongue are not affected. Hypertrophy of the tongue, or *macroglossia* is generally congenital, but may occur late in life. The tongue enlarges, and is accompanied by pressure symptoms, due to such enlargement. Hypertrophy of the tongue is sometimes seen in idiots and cretins. The hypertrophy is more frequently the result of lymphatic obstruction, on account of which there is lymph-stasis. The diagnosis is easy. Inflammatory hypertrophy occurs in stomatitis, and syphilitic hypertrophy occurs with gummata. Cysts. Various cysts occur in the tongue. The mucous cysts and blood cysts are the most common. The mucous cyst, the cysticercus cellulosæ, and the echinococcus occur rarely. Ranula is a cyst underneath the surface of the tongue that causes mechanical suffering. It is easy of recognition.

Parasitic Disease of the Tongue.—Thrush is the most common.

The Effects of General or Remote Disease on the Tongue. *The Coating.* With a view to estimating the condition of the system in general from the appearances of the tongue, excluding all local conditions, the following characteristics are observed: first, the color; second, the fur; third, the degree of moisture; and fourth, the movements. The student should bear in mind that changes in the condition of the tongue are frequently of local origin; that dryness, for instance, may be due to the open mouth, or that a coating may be unusually marked because the tongue had not been used in mastication. Often coating is seen on one side of the tongue. This has been referred to as due to disease of the nerve of one side. It is just as likely due to an absence of mastication on that side of the mouth, the bolus of food being kept on the other side because of pain, diseased teeth, or other local cause.

Clinical experience has shown that certain states in the tongue are associated with certain general conditions which render the appearance somewhat diagnostic. The term diagnostic must be qualified because of the fact that the changes are so often local, or that they are modified by conditions independent of the general system. For convenience, the classification of Dickinson as to the appearance of the tongue in disease may be utilized. In the Lumleian lectures this eminent authority described the average healthy tongue based on extensive observations. Departures from the normal were arranged and afterward classified. It resulted in the formation of eleven classes. The first was the *stippled* or *dotted tongue.* The tongue was moist and dotted with little white points, due to an excess of white epithelium on the papillæ. It is usually seen in persons in poor health without fever. It is not, therefore, a febrile tongue, nor one indicative of grave constitutional disease. It is seen in cases of chronic disease, usually in which there were no grave symptoms. Second, when dryness attends the stippled tongue it is found in mild acute diseases, or in cases in which the constitutional disturbance is more marked. The third class is *stippled and coated.* The patients in whom this is found very frequently are the subjects of acute and constitutional affections. Fever is more frequently present in cases of this fur. Fourth, the *coated tongue.* There is excess of white epithelium on the papillæ, and the coat is continuous. The intervals between the papillæ are filled up with epithelium and accidental matters more commonly than in the preceding. It is seen in the acute and febrile diseases. In the moist and dry kinds, pneumonia, pleurisy, typhoid fever, and other febrile disorders make up the list. Prostration and pyrexia attend the cases in a far greater degree than the preceding, while the saliva is absent in a larger proportion of the cases.

The Strawberry Tongue. The tongue is coated and injected, the fungiform papillæ shine through the coat, particularly at the tip and edges. It is the tongue of scarlet fever, but may be seen in any acute febrile disorder. Pyrexia is more common in this class than in the preceding.

The Plaster Tongue. A thick, uniform coat, abrupt and striking, covers the tongue. The papillæ are elongated and the intervals crowded with accumulations, among which are bacteria; it is the tongue of acute febrile disease. Fever was marked in a number of cases Dickinson studied, and prostration was a common attendant. Saliva was deficient. It is thus seen that, beginning with the healthy tongue, Dickinson described a series of groups, in each succeeding one the coating becoming more marked, with or without moisture. The clinical association that he found is a common experience. Each successive group was attended by more fever and greater exhaustion and less saliva than the preceding group, and in each the tongue became more and more furred.

The Furred or Shaggy Tongue. When moist, the papillæ are greatly elongated, composed mostly of horny epithelium. It has the same appearance as if the tongue was dry. The moist, furred tongue is not as common as the other. It is most commonly seen in old age and in constipation. The dry, furred, or shaggy tongue may succeed the dotted tongue or the coated tongue in the course of advancing disease. It is the result of disease and want of moisture. The saliva is deficient; there has been fever and possibly but little food used.

The Incrusted, Dry Brown Tongue. Over the surface of the tongue there is a dry, thick, felted coat, which is continuous and dips down between the papillæ. The coat is largely made up of parasitic material. In the course of fevers it is the outcome of a preceding condition, the coated tongue, and is indicative of the typhoid state. It occurs in the fevers with high temperature, but may be seen in conditions of low temperature, as from cancer, phthisis, albuminuria, chronic nervous diseases. There is much depression or prostration associated with it, and there is absence of saliva. If the patients with a dry brown tongue recover, it retrogresses to the furred or incrusted tongue, which in turn becomes bare gradually, at first in small layers; it is thin, usually dry, but is more moist than the dry brown tongue As the incrustation disappears it may become bare, red, and dry. The *red dry tongue* indicates a more serious condition usually than the dry and brown. It is the tongue of chronic wasting diseases. It occurs in phthisis in the later stages, and, as the raw-beef tongue, is associated with dysentery and also with liver abscess. There may be fever associated with the cases. It is in a measure the tongue of chronic diarrhœa, and particularly the form known as tropical diarrhœa. The tongue is shrunken, red, polished, and smooth. The papillæ have disappeared and the epithelium stripped off in patches. It may be associated with aphthæ. If the patient is to improve, the redness fades, the papillæ become softer, and the moisture returns.

Cyanosis, or Venous Congestion of the Tongue. The tongue is of a bluish or purplish color, the surface is smooth and wet, and the papillæ are almost indistinguishable. It is not confined to organic heart disease or cyanosis. It is of quite frequent occurrence in albuminuria. With the venous congestion in the albuminuric cases there is always a superabundance of deep epithelium. When the surface is examined it looks as though the papillæ were fused together, over which may be laid a moderate coat.

Classification of Tongues.

To the naked eye.	*Microscopically.*
1. Healthy, moist.	White epithelium in small amount on papillæ, not continuous or superabundant.
2. Stippled, moist, dotted with white.	Excess of white epithelium on papillæ, not extending between them.
2 (D)[1]. Stippled, dry.	Ditto.
3. Stippled + coated; moist. Coat continuous in parts.	White epithelium on papillæ in excess with partial filling of intervals.
4. Coated white; moist. Coat continuous.	Excess of white epithelium in papillæ. Intervals more or less filled up with epithelium and accidental matters.
4 (D). Coated white, dry. Coat continuous.	Ditto.
5. Strawberry, coated + injected, especially showing in fungiform papillæ	Like the coated or plastered, but with more injection.
6. White, plastered, thick, uniform coat, abrupt and striking.	More elongation of papillæ than with coated tongue, more filling of intervals with superficial accumulation.
7. Furred or shaggy, moist. Greatly elongated papillæ.	Extravagantly long papillæ, mostly of horny epithelium.
7 (D). Furred or shaggy, dry.	Ditto.
8. Incrusted, dry, brown; thick, felted dry coat over papillæ.	Continuous crust on and between papillæ, largely of parasitic matters.
9. Furred or incrusted, becoming bare. Generally dry.	Crust breaking away, together with more or less of normal surface.
10. Denuded, red. Absence of normal covering.	General absence of all epithelium excepting the Malpighian layer; sometimes of that also.
11. Red, smooth, dry, membranous covering.	Level membrane replacing epithelial processes.
12. Cyanosed.	Injected; hypernucleated; excess of deep epithelium.

Moisture of the Tongue. The moisture is due to the saliva generally, any deficiency of which causes dryness of the tongue. It is natural, therefore, to appreciate that any changes in the moisture of the tongue are due to the secretion of the salivary glands. Fever is almost always present when this is deficient, and hence the tongue is dry. At the same time this failure of secretion of the salivary glands does not attend diminished secretions elsewhere, unless it should be the glands of the gastro-intestinal tract.

Dryness of the tongue, it must not be forgotten, may be due to increase of evaporation due to exposure of the mouth by persistent

[1] The letter D is used to imply dryness. Thus, to Class 2 a certain description is attached. Class 2 D presents the same characteristics with the addition of dryness.

openness, in addition to diminution of the salivary secretion. All states, therefore, in which the mouth is open will lead to dryness of the tongue. Again, in chronic fever, dryness of the tongue is a constant characteristic. Dryness is due to the effects of the temperature upon the secretions in general, but it is not the effect of high temperature, curiously, but rather a temperature which has persisted for a considerable length of time. Thus, in pneumonia, with a temperature of 105,° the tongue may be moist; whereas, in typhoid fever, with a temperature of 103,° the tongue is dry. General dehydration of the body causes dryness of the tongue, even without diminution. This dehydration is seen in diarrhœa, in which disease simple or uncomplicated dryness of the tongue is a common symptom. It is curious to observe that in cholera the tongue remains moist even until death; whereas, if the patient is about to improve and the discharges cease, reaction and fever setting in, the tongue begins to dry and becomes quite brown. Local causes may explain this. The watery vomit may keep the tongue moist, and the temperature of the body may contribute to the change. Next after diarrhœa we find excessive discharge of urine the cause of dryness. Hence, in diabetes in all forms, extreme dryness of the tongue is seen. The osmotic action of the sugar in the blood is the cause of a reaction in diabetes mellitus, just as it is in cases of dehydration of the lens in cataract. The final cause of dryness of the tongue is prostration. Asthenia in all forms which continues over a moderate period of time, as a week or ten days, causes lingual dryness.

The Effects of Food. These must be studied before deciding upon the clinical significance of changes in the tongue. The immediate results of food have influence in determining the coating and the degree of moisture. The act of eating cleans the tongue. In disease, therefore, in which this act is not performed, it is natural that we observe more fur on the surface, and in conditions in which diet is limited to fluids the effect is marked. In cases of liquid diet, the tongue is liable to remain furred. It is particularly seen in patients who are kept upon a milk diet exclusively.

The Tongue in Relation to Diseases of the Alimentary Canal. So much has been written on this sbbject that it is well to give the experience of Dickinson briefly. He declares that he has not been able to discern any relationship between any state of the tongue and dyspepsia and ulcer of the stomach apart from that which might occur from loss of appetite or limitation of the food. With regard to the bowels, some forms of constipation are often connected with changes in the tongue, but such connection is not necessary. The author rather thinks it to have been a coincidence, and cannot even point to the diagnostic significance of the tongue in obstruction. The state of the tongue in the latter condition is dependent, not upon the intestinal lesion, but upon the constitutional disturbance. A dry tongue is well known to occur in acute obstruction. He thinks that this is due to deficiency of salivary secretion; unless, however, there is constitutional disturbance, he does not think that in chronic obstruction the tongue will change. In diarrhœa all conditions of dryness, furring, and incrustation are osberved. The absence of saliva, dehydration, and pyrexia help the

desiccation. In diarrhœa and dysentery, therefore, the change in the appearance of the tongue is more marked than in any other disease.

Other Diseases. In relation to other individual diseases but little may be said. Of more direct association, we have the tongue of heart disease, of which the cyanotic character is evident, a similar condition sometimes accompanying chronic albuminuria and diabetes mellitus, in which there is excessive dryness; the tongue of scarlet fever and of typhoid fever have been referred to, the strawberry tongue of the former being almost pathognomonic. Of course the so-called typhoid tongue represents but one stage of typhoid fever. Throughout the disease it may present all varieties in direct succession, from the stippled, the coated, the plastered, the furred, to the incrusted. In lobar pneumonia the same changes occur as the disease advances. In bronchitis the lower degrees of coating are presented, while in rheumatism the variety is considerable. In conclusion, it may be stated that the tongue seldom points to solitary organs or isolated disorders, but is a gauge of the effects of disease upon the system.

The Tongue in Treatment and Prognosis. Clinical observers agree with Dickinson that the condition of the tongue is due very largely to the four states with which he has associated it—dehydration, exhaustion, pyrexia, and local conditions about the mouth. As these conditions cause the state of the tongue, it is evident that the first sign of its improvement, as return of moisture, denotes a diminution in temperature. Its appearance is, therefore, of good prognostic omen. The condition of fever, the state of the nervous system, the maintenance or abeyance of secretions, and failure of vitality, are indicated by the condition of the tongue. The return of moisture, the removal of fur, the subsidence of tremor, at once indicate that the patient is getting better. The persistence and increase of these signs show that the disease is getting the better of the patient. As to indications for treatment, the dryness, furring, and incrustation are connected with the want of saliva. The processes by which this want is brought about differ. They have previously been referred to, and the indications for treatment are obvious. The inference from the state of the saliva as to the condition of the intestinal canal is of the highest importance practically. There is no doubt that, except in diabetes, when there is diminished saliva, there is also diminished gastro-intestinal secretion. Such diminution is followed by loss of appetite and impairment of digestion. The indication is to at once administer material that is digested with the least difficulty. Hence liquid food and stimulants are to be used. The dry and bare tongue is of serious prognostic omen in all conditions. While it may be due to want of saliva alone, it also occurs as a part of the failure of nutrition in hectic fever, suppuration, and other conditions. It is an indication for the use of tonics, stimulants, and food, probably liquid, always nourishing. The weak pulse does not more surely tell of asthenic tendencies than the red, dry, and polished tongue.

Movements of the Tongue. When the patient is asked to put out his tongue in health he complies without any undue movement save that required for its ejection. In general states, or in disease which has caused an interference with its motility, the projection is attended by

abnormal movement. It may be tremulous, as in alcoholism or weakness. It may be slow or impeded in the stages of various paralyses. It is tremulous and the seat of fibrillar contractions in general paralysis. In glosso-labial paralysis the tongue cannot be projected at all. In general paralysis and diphtheritic paralysis, progressive muscular atrophy and hemiplegia, the paralysis is only partial, and hence, while projected, it is done with difficulty, and may have to be aided by the finger. In hemiplegia, in which the face is involved, the tongue points to the paralyzed side of the body.

Angina Ludovici. Angina Ludovici is characterized by slight inflammatory congestion of the throat out of proportion to the symptoms of the inflammation in the external structures. Woodeny induration, which will not receive impressions, of the connective tissue, spreading of this induration instead of fading off, so that it is bound sharply by unaffected cellular tissue, may extend from the rami of the jaws to the face. With this there is a hard swelling in the *tongue* and along the interior lower jaw, causing thickening of the floor of the mouth. This is observed by palpation with the finger in the mouth. The glands are not affected. For a long time the nature of this affection was not known. It is now believed to be due to actinomyces. (See Parker, *Lancet*, 1879, and Anderson, *Transactions of Medico-Chirurgical Society*, 1891.)

The Fauces and Pharynx.

The passage-way between the mouth and the respiratory passages is lined with mucous membrane, which is subject to diseases to which they are liable. The symptoms thereof are similar to the symptoms of mucous membrane inflammation elsewhere. The large muscles of the pharynx which aid in deglutition are subject to affections which belong to muscular tissue generally, hence *rheumatic inflammation* and loss of power of the muscle, or *paralysis*, occurs. Situated in the position that the pharynx is, it is particularly liable to *infection* from micro-organisms. The infection may extend from the mouth, or above from the nares, or the micro-organisms may affect this locality primarily. As a passage-way or channel, the affections of the pharynx are liable to occlude it, on account of which symptoms arise due to the *occlusion*. In addition to its function as a simple channel, the pharynx is concerned in the act of deglutition. When, therefore, there is obstruction of the pharynx, *deglutition* is made difficult, or even may become impossible. As a channel for the passage of air, obstruction in the pharynx will lead to *dyspnœa*.

The fauces and pharynx may be the seat of morbid processes which occur secondarily to diseases in other portions of the body with a moderate degree of frequency. It is true that inflammations of the mucous membrane of the pharynx have to bear the blame of rheumatic or gouty origin in a large number of cases, according to the opinion of many observers. Indeed, gouty inflammation of the pharynx seems to be more common than gouty inflammations of mucous mem-

branes in other situations. In the large majority of pharyngeal inflammations that are subacute or chronic they are secondary to dyspepsia. The *secondary* processes occur chiefly from extension of the disease in the cavities related to the pharynx, and are not of special diagnostic significance. The following pharyngeal diseases point to primary conditions, general or localized, elsewhere.

Paralysis of the pharynx does not have the diagnostic significance of central lesions that paralysis of other structures, as parts of the larynx, have. This is due to the fact that the nerve supply of the pharynx is derived from a nerve (glosso-pharyngeal) which supplies other structures, paralysis of which is more readily ascertained, and which causes more pronounced symptoms. (See Cerebral Nerves.) Affections of the tonsils are usually more common in rheumatic states, and bear some relationship to the rheumatic diathesis. Inflammation of the tonsils may follow acute rheumatism or may alternate with it. A patient who is predisposed to rheumatism may at one season have tonsillar inflammation, at another rheumatism. The writer has seen tonsillitis immediately followed by rheumatism.

Apart from what has just been said, diseases of the pharynx bear but little if any diagnostic relationship to disease elsewhere. While there may be cyanosis of the mucous membrane, or tuberculous ulceration, or other changes which we have noted, the signs of the primary disease are so much more marked coincidently in other situations that we need not rely upon the appearances of the pharynx or symptoms of pharyngeal disease for diagnostic purposes. The only general affection which may be diagnosticated from the appearance of the pharynx alone, is measles. In obscure cases of sudden fever, with nasal catarrh, the appearance of the eruption in the situation previously indicated may lead to the recognition of measles when the external eruption is not characteristic. For the purposes of the therapeutist it should be borne in mind that symptoms referable to the pharynx are very frequently due to disease in the nares, and particularly in that portion of the pharynx which is not open to direct inspection—the naso-pharynx.

The *general symptoms* of pharyngeal disease are not marked, except in diphtheria, in erysipelas, in retro-pharyngeal abscess, and in affections of the tonsils. In the latter the general symptoms appear to be out of proportion to the local process. The high fever, the intense headache and backache, and rapid pulse, point to a process which in extent and severity should far surpass that which occurs in the tonsils.

Attention cannot be too strongly directed to the investigation of the naso-pharynx in children who are poorly developed physically, in whom there is backward mental development, and who present appearances that, to the practised eye, are most familiar. The experienced observer will at once judge, and judge correctly, that this combination of symptoms is due to disease in the naso-pharynx. Reference must be made to the remarks on adenoid vegetations of the naso-pharynx, but it is proper to state here the relationship and the importance of investigating the structures in the class of cases just indicated.

The Objective Symptoms.

The objective symptoms are noted by inspection of the pharynx.

The Examination of the Fauces. For this purpose examination is made by the unaided eye, but with the part illuminated in the usual manner. The difficulties arise from the tongue and the uvula. The mouth should be opened in a relaxed manner, not so wide as to be a strain upon the patient, but as wide as is consistent with comfort. The tongue is pressed down out of the way by the use of a tongue depressor. In many cases, however, even with the tongue depressor, the tongue muscles will contract and the organ bunch up in the mouth. Moderate, quiet, full breathing, gently opening the mouth as the deeper inspirations are made, causes the tongue to be relaxed and lie in the bottom of the mouth, and at the same time elevates the uvula. At the time of the full breath the part may be inspected throughout. Sometimes the fauces can be examined if the tongue is protruded and held between the finger and thumb of the patient with a soft napkin. In the fauces the tonsils and uvula are to be observed, following out the routine method of ascertaining all facts. Attention is then paid to the posterior wall of the pharynx with the same object in view.

Method. On examination of the fauces and pharynx, observation is made of the *color* of the parts, the appearance of the mucous membrane and its glands, the *appearance* and *position* of the uvula, the size of the tonsils, the character of the secretions on the pharynx, and the presence or absence of swellings and abnormal exudations.

Color. The color of the mucous membranes generally is of a dark red hue. The color is increased in intensity in acute inflammations of the pharynx, whether primary or secondary. In the acute forms it is bright red in color. In cases of heart disease, when there is cyanosis, the veins are congested. In obstruction of the superior vena cava by tumor there is similar change in hue of the surface of the pharynx. The capillary vessels may pulsate in aortic regurgitation. Bleeding-points may be seen over the surface of the pharynx, which may give rise to hemorrhage to such a degree as to simulate pulmonary hemorrhage. The blood may be swallowed and then vomited and the patient be thought to have a gastric hemorrhage. When the hemorrhage occurs at night it is seen on the pillow as yellowish stains. It is often due to adenoid vegetations in the naso-pharynx.

On examination of the posterior wall of the healthy pharynx little elevations due to glands are seen upon its surface, and moderately sized vessels are seen coursing through the mucous membrane.

Eruptions. Eruptions may be observed in the pharynx in some of the specific fevers. Thus, in measles, the appearance of the rash on the pharynx and the soft palate may be observed before the development of the rash on the surface of the skin. The eruption of scarlatina is also seen in the pharynx, and the papules and pustules of variola are frequently observed in that affection.

The Tonsils. The tonsils are situated at the sides of the pharynx between the anterior and posterior folds of the palate. They are pathologically of much importance. They are made up of glandular

structure arranged in follicles and held together by connective tissue. The crypts of the follicles open on the surface, and in disease are visible. The tonsils are small bodies, not larger than a filbert in the adult. Their entire surface can usually be seen by ordinary inspection. If enlarged the posterior surface cannot be seen, although a larger view may be obtained by causing the patient to gag or retch, during which they are brought forward to the light. The diseases of the tonsils do not have any relationship to their function as far as known. The tissue and gland follicles are liable to inflammations, which may be bacterial or may be the result of rheumatism. The tonsils become enlarged; the swelling takes place rapidly in the acute forms. They may be simply enlarged and the covering membrane intensely red. In other forms of inflammation the surface may be dotted over with white points, due to exudation from the follicles, which may be covered with a white or grayish membrane, which is removed with difficulty, leaving an abraded face underneath. Repeated attacks of inflammation cause chronic enlargement of the tonsils. They are enlarged sometimes to a great degree, filling almost entirely the lumen of the fauces. The surface is irregular, and may be scarred. The mouths of the follicles may be dilated. By virtue of their position, enlarged tonsils from any cause are the source of dyspnœa and dysphagia. The tonsils may be the seat of sarcoma.

The Uvula. In health it hangs midway from the palate. It varies in shape from congenital causes, and may be elongated on account of disease. This particularly takes place if there has been hawking or coughing on account of chronic nasal catarrh. When elongated it is pointed and may extend almost to the base of the tongue. The uvula may be swollen and œdematous. The œdema is usually associated with subcutaneous œdema in the course of Bright's disease. It may occur in debility. In both conditions it may become so enlarged as to interfere with swallowing and breathing. In some cases of pharyngitis the uvula is the seat of intense inflammation and great œdema. In addition to the constant cough which it causes there may be dyspnœa and repeated attacks of strangulation.

Hemorrhagic infarcts may take place in the uvula. In two instances under the writer's care the intense infarction led to sloughing, and in one the uvula was swallowed.

Ulceration. Follicular Ulceration. Small superficial ulcers corresponding to the follicles may be seen over the posterior wall of the pharynx. They occur in chronic catarrh, and are due to the inflammation of the follicles. In addition, ulcers secondary to infectious processes are sometimes seen, as in *typhoid fever.* In *syphilis,* in the secondary stage, small, shallow ulcers are seen, on the posterior wall of the pharynx. They do not cause pain. Mucous patches are observed at the same time, not only on the pharynx, but also in the mouth. In the tertiary stage, deep ulcers, followed by scars, are seen on the posterior wall of the pharynx. Although the absence of pain renders it probable that they are of syphilitic origin, nevertheless the history of infection and of the primary lesion, and the evidence of the disease in other structures may be secured before diagnosis is fully established.

In the tertiary forms it may be necessary to resort to the therapeutic test.

Tuberculous ulcers are irregular and the floor grayish. They are seen in tuberculosis in its later stages. They are the source of extreme pain. There is usually ulceration in the larynx at the same time, and, in extremely rare cases, tuberculous ulceration of the tonsils. In a patient, a lad of sixteen, under the writer's care, the large tonsils were of a honeycombed appearance on account of the grayish, irregular ulceration. Deglutition was absolutely impossible on account of the pain, and the young man died of starvation. In tuberculous ulceration, after the application of cocaine, a portion may be scraped off, and a microscopical examination will show the presence of bacilli.

Cancer of the pharynx is rare, and is usually secondary. The disease has advanced from other situations.

Exudations on the tonsils are due to inflammation of the follicles, to diphtheria, to the pseudo-diphtheritic inflammation which attends scarlatina, or which arises secondarily to other infectious debilitating diseases, and to thrush. On the pharynx the exudation may be due to diphtheria, to pseudo-diphtheria, or to thrush. The method of distinguishing the various forms will be considered in the articles on the respective affections just mentioned. In diphtheria the membrane is made up of fibrin arranged in a network, in the meshes of which epithelium, blood and pus corpuscles and micro-organisms are found. When removed, hemorrhagic abrasions and raw purulent inflammation remain. The two forms of bacilli are found in the membrane; the pseudo-diphtheritic bacillus or streptococcus, and the true, or Löffler's bacillus (see Bacteriology). The Löffler bacillus is best detected by cultivations. After the membrane is removed with the usual antiseptic precautions and washed in a 2 per cent. solution of boric acid, it is cultivated in blood-serum.

The pseudo-diphtheritic bacillus likewise grows, but its appearances are different.

Anæsthesia. In addition to the evidences of pharyngeal disease, observed on inspection by means of the probe, alterations in the sensibility of the pharynx may be detected. In the whole posterior wall of the pharynx sensation may be absent. This may occur in hysteria, in bulbar paralysis, and in diphtheritic paralysis. On the other hand, there may be an apparent *hyperæsthesia.* In some individuals the pharynx is particularly sensitive to the presence of foreign bodies, as inflammatory exudates, and may resent their presence by sudden coughing and retching. Inflammations increase the hyperæsthesia of the pharynx, and it is sometimes observed in hysteria.

The cervical glands. The pharynx is in such intimate connection with the large lymphatic glands in the neck that diseases of the former are frequently attended by enlargement of the latter. The glands that are enlarged are situated at the angle of the jaw. The lymphatics extending down the neck along the vessels may also be enlarged. In cases, therefore, of enlargement of the glands in this situation it is absolutely essential to examine the fauces and pharynx.

Leptothrix of the Tonsils. In healthy persons the plugs which block

the tonsillar crypts are found to be made up of cells and segmented fungi. The latter stain bluish-red with the iodo-potassic iodide solution. Sometimes the micro-organisms extend beyond the follicles, covering the surface of the tonsils with patches of various size. They are thus seen in follicular tonsillitis.

Subjective Symptoms.

Pain. In affections of the fauces and pharynx pain is one of the most common subjective symptoms. It is due to the fact that the functional acts of the pharynx require movement of all the structures. When they are the seat of inflammation, or ulceration, the movement excites pain. It is, therefore, an intense symptom of inflammation of the tonsils and pharynx, of rheumatism of the muscular structure of the pharynx, and of tuberculosis and cancerous ulceration. Pain in the pharynx is a frequent accompaniment of post-nasal inflammations, the pharynx not being the obvious seat of inflammation.

Dryness. Dryness of the fauces, with tickling sensation and a more or less constant desire to hawk, occurs in pharyngitis. Hawking, however, is not a symptom of disease of the pharynx alone. Its occurrence can only be explained, often, by disease in the posterior nares.

The Odor of the Breath. In follicular tonsillitis a peculiar odor is given to the breath. This is more marked in the milder grades of inflammation, with retention of the secretion of the glands. The odor is intense and fœtid. In cancer and syphilis there is also fœtor of the breath. The presence of the fœtor may be of diagnostic significance in the distinction of cancer from tuberculosis.

Dysphagia. The symptom varies in degree from slight difficulty in swallowing to complete prevention of the act. Any disease which occludes the passageway causes dysphagia, but it occurs independent of obstruction, on account of pain. It is, therefore, present in all painful affections. The pain causes the difficulty of deglutition. *Dyspnœa* is seen in tumors, in inflammation of the tonsils, in the rare form of erysipelas of the pharynx, and in retro-pharyngeal abscess. It occurs from occlusion of the passages, and is more marked in retro-pharyngeal abscess and erysipelas than in other conditions. In certain forms of abscess of the tonsils it may be very extreme.

Spasm of the pharynx is a subjective symptom that the patient complains of in some cases of intense pharyngitis. The degree of spasm or the amount of choking sensation is largely dependent upon the neurotic constitution of the individual. It may be extreme when only a moderate amount of inflammation is present. It is seen in the most aggravated form in cases of hydrophobia.

Tonsillitis.

Acute inflammation of the tonsils may be confined to the follicles, to which the term follicular tonsillitis is applied, or it may be limited to the mucous membrane, when it is known as catarrhal or erythematous tonsillitis. If with the catarrhal inflammations vesicles appear on the

mucous membrane of the surface, the term *herpetic tonsils* is applied to it. When the inflammation extends to the stroma of the glands it goes on to suppuration. It is characteristic of all forms of acute inflammation of this gland to recur frequently in the same subject. The relationship to rheumatism has been spoken of. This relationship applies in both the acute and the suppurative forms. All varieties may occur at any age, although it is least common under ten years of age; while the suppurative form occurs in adolescence. It occurs in both sexes. It may follow exposure to wet and cold, although patients who are subject to the attacks bear the exposure, unless at the same time they are unduly fatigued. The *follicular* form of tonsillitis is so frequently associated with bad drainage or other unhygienic conditions that emanations under these circumstances appear to be an exciting cause. Several persons of the same family may be affected at one time, which makes it often difficult to distinguish the cases from diphtheria. The disease, however, does not seem to be contagious. Persons brought in contact with the family, but who do not reside in the same house, escape the disease. This applies as well to children, who would, if the cases were diphtheritic, be more liable to become infected. The disease occurs more commonly in spring than in any other season of the year, but is generally noted during cold and wet seasons.

Symptoms. In follicular tonsillitis, with or without a rigor, but always with chilly sensations, the temperature rises rapidly to a great height. The subjective sensation of fever is very quickly noticeable to the patient, and more pronounced than in other affections generally. With the chill and during the rise of temperature there is some frontal headache, severe pain in the back and in the limbs. The pain in the back is most excruciating. In a short time the patient complains of the throat. Swallowing is difficult, and there is a sense of fulness. The throat is dry and burning. On examination the tonsils are swollen and creamy, and a yellowish-white exudation is seen on the crypts. The glands expand slightly, and may extend only slightly beyond the arches, or in younger subjects may extend one-quarter the way into the lumen of the fauces. Sometimes one gland is affected before the other. The difficulty in deglutition increases and the voice becomes nasal. There is usually some enlargement of the cervical glands. The general symptoms continue for forty-eight hours, the temperature remains at 105°, and the pulse is very rapid. After the first twenty-four hours the pain in the back lessens. The tongue is coated, the breath is heavy. The urine is loaded with urates. At the end of the fifth day the fever, which subsided gradually, has disappeared. The local symptoms, however, may remain longer. That is, the tonsils are enlarged and the exudation disappears slowly. Sometimes the prostration and general symptoms are very severe, so that after the fever has subsided convalescence may be very slow.

Albuminuria, due in all probability to the fever, frequently occurs; in some cases, undoubtedly acute nephritis attends the attack, and it is the cause for prolongation of the convalescence. In a case under the writer's care the patient first had acute rheumatism; this was replaced by a severe attack of tonsillitis, during which albuminuria, blood and

granular casts were found. The tonsils subsided in due course, but the Bright's disease continued during a long period, finally ending, however, in complete recovery.

Diagnosis. The diagnostic features of acute tonsillitis are the sudden high fever, severe backache and headache, pain in the throat, and albuminuria. The characteristic appearance of the face, the salivation and pain, with suppressed voice and impossible deglutition, should not cause it to be confounded with trismus or *tetanus*. In both, the jaws are closed. It must not be confounded with *smallpox*, which it resembles during the first twenty-four hours.

Cases of follicular tonsillitis are frequently mistaken for *diphtheria*. The inflammation in tonsillitis is limited to the glands, on which are patches of a yellowish-gray color, which are easily removed and do not leave bleeding surfaces. In diphtheria, the membrane is of an ashy-gray color, not in points or small patches, or separated by red tonsillar tissue. In diphtheria the membrane extends to the pillars of the fauces, and may appear on the uvula. There are, nevertheless, many cases which are doubtful, and bacteriological diagnosis must be resorted to (see Bacteriological Examination). The cases that particularly increase our anxiety are in adults who are subject to attacks of follicular tonsillitis. In addition to the results of the bacteriological examination stress must be placed upon the history of exposure. In the grave and extensive forms of diphtheria with asthenic symptoms the diagnosis is not difficult.

In *herpetic tonsillitis*, the severe pain and intense general symptoms are out of proportion to the local lesion. In *suppurative tonsillitis* the constitutional disturbance is also very great. The temperature rises high, 104° to 105°, and the pulse is very rapid, from 110 to 130 in the adult. The inflammation usually begins in one tonsil first. It may be limited to the one side, or the other be involved later. The tonsils at first are enlarged and firm and very red. There is swelling of the tissues around. In twenty-four hours deglutition is almost impossible, and there is salivation. At the end of forty-eight hours the patient presents a striking and distressing appearance. The glands of the neck are enlarged, the patient is unable to open the mouth, the voice is nasal or almost suppressed; there is dribbling of saliva from the mouth. The face may have a dusky hue in the midst of a capillary congestion due to the fever. There is constant desire to discharge saliva and accumulated secretions from the back part of the mouth. The patient cannot lie down. The pain is extreme, and is aggravated by swallowing. It is sometimes of a throbbing character and often shoots to the ears. Indeed, the earache may be the chief pain complained of. The patient does not take food, and exhaustion soon ensues. During the twenty-four hours before rupture takes place, on account of the exhaustion, the previously reddened face becomes blanched and dusky. The fever is continuous during this time, along with rapidity of the pulse. The patient may have been delirious. Sometimes the delirium is marked and the patient resists efforts to keep him in bed because of the intense discomfort of lying down.

The suffering is out of proportion to the danger of the case. About the fourth or fifth day suppuration has been completed, and if the finger

can be inserted into the mouth between the almost closed teeth, fluctuation is detected. In cases in which the mouth is opened a little more freely, in addition to the swelling of the tonsils below the arches, marked swelling and projection forward of the half-arches may be seen. The fluctuation may be detected through the anterior fold of the palate, and if lancing is to be performed pus can only be reached through this structure. After spontaneous rupture, which usually takes place upward into the mouth, instant relief is given. It may rupture into the pharynx, and suffocation may follow on account of the entrance of pus into the larynx. In rare cases it has opened into the carotid artery with the occurrence of sudden death from hemorrhage.

Enlargement of the Tonsils.

Chronic Tonsillitis. The tonsils may be enlarged on account of repeated attacks of acute inflammation, or attacks of chronic inflammation. They do not appear to be the cause of serious symptoms unless associated with adenoid vegetations in the naso-pharynx. They may interfere with hearing, however, and cause snoring at night. A fœtor of the breath may be noted, particularly if the secretion lodges in the crypts. This may be recognized by its characteristic yellowish color and by its odor on removal. The enlarged tonsils are irregularly formed and the surface is somewhat irregular.

Foreign bodies in the tonsils are not of common occurrence. They give rise to local symptoms, that is, to the sensation of the presence of a mass causing repeated efforts at swallowing. If calculi are present the patient may complain of a rough body. The calculi and rough sensations follow frequent attacks of quinsy. Hydatids are sometimes located in the tonsils.

Adenoid Vegetations of the Naso-pharynx.

While this name is applied to an abnormal increase of the tissue of the pharynx, other names have been given and various views held as to its occurrence. Some authorities have held that the vegetations were new growths, while others that it was simply a hypertrophy of the normal tonsillar tissue, the pharyngeal or third tonsil, which is situated in the locality in which they are found. The symptoms are due to stenosis of the pharynx, and are general as well as local.

The Nose. The nostrils are flattened laterally. Rarely they may be depressed. In one instance which the writer saw with Dr. Harrison Allen the exterior of the nose suggested inherited syphilis, all the more because of our knowledge of the possible occurrence of the disease. There were no other evidences of hereditary syphilis in the child or in any members of his family. In a large number of cases there is a discharge from the nose. This may be muco-purulent, or be associated with crusts. If the discharge is not constant the child is subject to colds and discharge on the slightest provocation. Independent of the chronic purulent nasal discharge mucus and blood may be passed at night and be found on the pillow in the morning.

The Mouth. The mouth is kept open, and there are evidences of mouth-breathing. The lips are always dry and may be cracked. They are thickened. The dental arch is high and narrowed.

The Voice. It is thick and muffled, becoming indistinct upon the occurrence of slight cold. The *expression of the face* is characteristic. It is dull and stupid, and may be drawn.

Mental and Nervous Symptoms. Headache, listlessness, and indisposition for mental exertion are marked. The patients are usually backward in their studies and are unable to fix their attention for any length of time upon any subject. *Aprosexia* is the term applied to this condition. The child is forgetful and cannot study without discomfort.

Choreiform spasm of the face occurs in connection with it. Enuresis is a frequent associate symptom. The child is subject to frequent attacks of indigestion. I have seen the following occur in many cases: Prior to operation the child had an abnormally poor appetite and was subject to frequent attacks of indigestion, characterized by vomiting, with fever. After the operation the appetite improved and continued good, and the attacks of indigestion disappeared entirely. The cases had been under observation before and after the operation for a number of years. The indigestion seems to have been due to the fact that on account of the obstruction the child would have to eat rapidly in order to keep the lumen of the mouth free for breathing purposes. The rapid eating, of course, prevented proper mouth digestion, and hence the occurrence of gastric catarrh.

Symptoms of Stenosis. In addition to mouth-breathing, the patient snores at night, and sleep is always disturbed. The respirations are irregular, with a pause between, followed by noisy inspirations. The difficulty of breathing is the cause of restlessness, and the child will often waken up in the night short of breath.

Night restlessness with *dyspnœa* and irregular respiration always point, therefore, to obstruction in the naso-pharynx. The *hearing* is frequently impaired. There may be simply dulness of hearing, or it may amount to marked deafness, either because of pressure of the adenoid vegetations or extension of secondary inflammation to the Eustachian tubes; the sense of taste and smell are often much impaired. There is increase in the secretion of pharyngeal mucus, which in older persons causes difficult expectoration.

The Appearances of the Chest While there is general lack of physical development, the physical development of the chest is most striking. The cases have been frequently mistaken for rickets, but in this country adenoid vegetations are a common cause of chest deformity; whereas, in England and Europe rickets is the most frequent cause. The ribs are prominent in front, the sternum is angulated forward at the manubrio-gladiolar junction and grooved at the gladiolar-xiphoid junction. A saucer-shaped depression is found at the lower costal cartilages. The ribs behind are closely compressed together, so that the intercostal spaces at the lower part of the chest are obliterated. The chicken-breast appearance is most striking, with the depression in the lower portions of the chest. The diaphragm may be drawn in during inspiration in the middle and lateral thoracic regions.

Rhinoscopic Examination. The floor of the pharynx is covered with rounded or villous projections, on account of which the posterior nares are often concealed. Rarely the villi may be seen projecting below the soft palate. In children the examination is difficult, and hence digital exploration must be used. This should be done under an anæsthetic unless there is no doubt whatever. The finger readily detects the masses, which sometimes are soft, at other times tough and of fibrinous or cartilaginous consistency.

The student cannot become too familiar with the symptoms and signs of adenoid disease of the naso-pharynx. There is no doubt that in cities of this country particularly this local affection is of more common occurrence and more disastrous in its results than any other local affection that we have to deal with in children. It may be said that in children in poor health, anæmic, with impaired digestion, and lack of muscular and physical development, if the causes are not due to impure air and improper diet or to improper sanitation, it may be almost certain that there is disease of the naso-pharynx. The writer has seen a very large number of cases in recent years in his practice, many of whom have been operated on by Dr. Harrison Allen, and has had the satisfaction of seeing the entire picture of the child change after proper treatment. It may be said in passing that this change does not take place at once, but after three to twelve months the child will be fully restored in physique, if during that time attention is paid to proper exercise and the development of the chest. Notwithstanding all this, however, the natural shape of the chest and appearance of the face are only gradually resumed.

Inflammations of the Pharynx.

Inflammation of the pharynx, *acute pharyngitis*, or sore-throat, follows cold or exposure, particularly after patients have been physically depressed; the inflammation often involves the tonsils as well as the pharynx. The symptoms are pain on swallowing, with dryness and a constant desire to hawk and cough on account of the tickling sensation. There may be slight laryngitis and inflammation of the Eustachian tubes, with deafness. Stiffness of the neck and enlargement of the cervical glands attend the local inflammation. The general symptoms are not marked. The attack is ushered in with chilliness and slight fever. On examination the mucous membrane is seen to be congested, dry, and glistening, and covered with sticky secretion in spots. The uvula may be very much swollen. The acute inflammation may be associated with rheumatism or gout. When the submucous tissues are involved the parts are more swollen and there is greater dyspnœa. The dysphagia is more marked, although the pain is not any greater. The larynx is always involved under these circumstances. The fever is higher.

Phlegmonous Inflammation. A diffused inflammation of this character occurs. The writer saw one case of this character so intense, with dyspnœa and high temperature, as to simulate pneumonia. Pneumonia was thought to be present because of the occurrence of congestion and

œdema of the lungs. It occurred during the prevalence of the recent epidemic of influenza. The disease began in the pharynx; the tissues were swollen and infiltrated. The early symptoms were pharyngeal. The dysphagia was extreme, and there was an abundant muco-purulent expectoration, which did not contain pneumococci. Death took place from exhaustion. The autopsy showed a high degree of congestion of the lungs, œdematous inflammation of the pharynx, larynx, and trachea. While, therefore, the recognition of an acute phlegmonous inflammation is not difficult it must not be forgotten that it is a grave disease which may terminate in such marked pulmonary symptoms as to lead to the suspicion of pneumonia.

Angina Ludovici is an inflammation of the cellular tissue of the floor of the mouth and the neck. It is probably a form of actinomycosis. The swelling is most marked below the jaw of one side. The symptoms are very intense and both local and general. There are general septic symptoms at once. With the swelling there is œdema and board-like induration. Redness and the rapid formation of an abscess rarely occur. The throat is not affected. Death takes place from reflex suffocation or in coma (see page 449).

Rheumatic Pharyngitis is of short duration, without objective symptoms. Pain is intense, deglutition difficult. The usual concomitants of rheumatism are present. It frequently gives place to torticollis, lumbago, or rheumatism in some other situation.

Chronic Pharyngitis follows acute attacks, or is a frequent accompaniment of nasal catarrh. It is common in smokers and alcoholic subjects; the use of the voice in loud tones, as by clergymen, auctioneers, etc., is also a cause. It is a frequent attendant upon indigestion, due probably to the eructations. The objective signs are relaxation of the mucous membrane, with dilatation of the veins. The membrane is covered with a thick secretion, which is dry and glistening. In the granular form the wall of the pharynx is covered with millet-seed projections and is congested. Tough mucus is seen in small areas.

Retro-pharyngeal Abscess. The inflammation may begin in the submucous connective tissue, and retro-pharyngeal abscess form. Dysphagia with stiffness of the neck and enlarged glands, with high fever, are present. On examination a projection into the pharynx can be seen or distinctly felt on the posterior wall. The disease may be difficult of recognition in infants, in whom it is impossible to get a good view of the pharynx. On the other hand, it may be simulated by disease of the cervical vertebræ, in which there is also stiffness, difficulty in deglutition, and possibly a tumor also. It must not be forgotten that retro-pharyngeal abscess may result from caries of the cervical vertebræ. In children the abscess is attended with dyspnœa and alteration in the voice, so that laryngeal disease may be suspected. I recall a case of retro-pharyngeal abscess in which the dyspnœa was so severe as to suggest croup, and indeed preparations for tracheotomy were made, when sudden rupture of the abscess revealed the nature of the disease. Fortunately the child had been kept in the upright position, on account of which the discharge of pus came forward to the mouth, or else suffocation would have ensued.

Inflammation of the Parotid Gland.

First, specific inflammation or parotitis (see Mumps); second, symptomatic parotitis, occurs in typhoid fever, pneumonia, pyæmia, and septicæmia. The process is intense, characterized by swelling, redness, and heat over the parotid gland. There are pain and difficulty of mastication; suppuration rapidly ensues. It is thought to be an unfavorable symptom, but I have seen two cases in typhoid fever get well. In a case of septicæmia it did not advance to suppuration. Stephen Paget has described a symptomatic inflammation in disease of the abdomen and pelvis He collected 101 cases, 50 of which were due to injury, disease, or temporary derangement of the genital organs, as by slight blows; or in females, the introduction of a pessary. It may occur before the menstrual period or during pregnancy. Septicæmia or pyæmia does not attend the process—indeed, many of the cases are afebrile. In 78 cases, 45 suppurated and 33 resolved without suppuration.

Gowers describes a case of parotitis which occurred in the course of fatal peripheral neuritis.

The Œsophagus.

The œsophagus is open to all affections which arise in mucous membranes, although its histological structure, position, and functions protect it from frequent involvement in disease. Should morbid processes arise, the symptoms expressive of these processes are the common symptoms of disease of the mucous membrane. But the œsophagus is a closed tube, the function of which is to afford entrance to and to propel food onward into the stomach. It is subjected to all the affections common to channels. Any disease of the tube interferes with its function, made evident by the symptom common to all disorders of the œsophagus —*dysphagia*. As this symptom occupies a position of such prominence in the symptomatology of diseases of this tube, it is evident that diagnosis of disease resolves itself into the differentiation of all forms of difficulty of deglutition.

Before beginning the discussion along the lines indicated, the subjective and objective symptoms of diseases of the œsophagus must be considered.

The Subjective Symptoms. *Pain* is a common symptom of disease of the œsophagus. In acute inflammation it is extreme, and is complained of in the neck, between the shoulders, and along the vertebræ for a short distance. Its character depends upon the cause. Severe burning pain, often agonizing, is due to inflammation from burns or caustics. After the ingestion of caustics the absence of pain points to extreme corrosive action and gangrene. Pain attends and is part of the symptom—*dysphagia* (*q. v.*). *Cough* attends such diseases of the œsophagus as exert pressure upon the bronchus, as carcinoma.

The Objective Symptoms. *Stiffness* of the neck is seen in acute inflammation of the œsophagus and in peri-œsophageal abscess; it also may occur in traumatism. The *expectoration* in diseases of the œsophagus is characteristic. It is usually a glairy mucus, often frothy

or viscid. It is not coughed up, but after welling into the pharynx is hawked up. It is abundant in acute and chronic inflammation and in cancer.

Hemorrhage from the Œsophagus. Hemorrhage from the œsophagus occurs from varicosity of the veins at the lower portion of the gullet. It may occur in old people, due to senile disease of the liver, kidney, and spleen, or arise as a complication in cirrhosis of the liver. In hemorrhage from the œsophagus the blood is usually bright in color, has not been acted on by the acid, and is not discharged by vomiting, although vomiting may occur after the blood is poured out. In a grave case of purpura under the care of the writer, hemorrhage took place from the lower end of the œsophagus. To distinguish it from gastric hemorrhage the stomach may be washed out. If this is done shortly after the hemorrhage by the introduction of a soft bougie, clear fluid will be discharged if the gastric mucous membrane is intact. Small bleedings from the œsophagus are usually indicative of cancer, if, in addition to the hemorrhage, there are present the symptoms of occlusion. Of the general symptoms due to œsophageal disease *emaciation* is the most characteristic. It is, of course, more striking in cancer, but occurs to a moderate degree in all forms of stricture. *Fœtor of the Breath.* This usually attends dilatation of the œsophagus only.

Emphysema of the subcutaneous connective tissue should always lead to the investigation of the œsophagus. Usually there have been pronounced symptoms of disease of the œsophagus. At times in rare cases ulceration may have gone on without symptoms. The ulceration, of course, extends into the air-passages with the occurrence of emphysema secondarily.

Physical Examination. Examination of the œsophagus is made by inspection and auscultation, and by means of palpation with or without a bougie. The œsophagus behind the trachea in the neck may be palpated when it is enlarged as in abscess. Palpation yields the most positive results. Inspection can be utilized only with an endoscope.

Auscultation of the œsophagus while the patient is swallowing fluids sometimes confirms the results obtained by instrumental palpation as to the seat of an obstruction. A gurgling sound is audible as the fluid passes the obstruction.

It must not be forgotten that the normal constriction of the œsophagus is present about opposite the fourth dorsal vertebræ, ten inches from the teeth. The bougie is of advantage in determining the cause of the difficulty in swallowing. If the cause is due to paralysis, or to spasm of the œsophagus the bougie can usually be passed with ease. If on the other hand it is due to organic disease obstruction will be found. The obstruction in organic disease is usually in the upper half of the œsophagus. Near the pharynx the obstruction is due to cicatricial stricture. Nine inches from the teeth, or about the position of the bronchus, the obstruction is usually due to cancer. The bougie should not under any circumstances be passed if there are strong grounds for believing there is an aneurism. Fatal rupture has followed its passage.

Method. The patient should be seated with the head thrown back sufficiently far to make the passage from the pharynx to the œsophagus

almost continuous. The operator may stand behind or in front of the patient. The bougie should be passed through the pharynx guided by the fingers and kept hugging the posterior wall of the pharynx. But little force should be used. It should be passed slowly and will soon overcome the gagging. The bougie should be warmed and oiled previous to being passed. The handles should be flexible, the bulb olive-shaped.

Dysphagia is a symptom common to all diseases of the œsophagus. It is seen in all forms of inflammation. It may amount to simple dysphagia on account of pain, or to the degree of complete obstruction of the tube. Dysphagia due to obstruction of the œsophagus is due (1) to disease outside of the canal, (2) to disease of the canal itself, and (3) to the presence of a foreign body in the canal. In the consideration of this symptom, therefore, these conditions have to be studied.

1. The œsophagus throughout its course is in intimate relationship with the trachea, the thyroid gland, the carotid artery, the left bronchus, the bronchial glands, the arch of the aorta, and descending aorta. Disease of these structures which admit of enlargement are liable, therefore, to cause difficulty in swallowing. It is not likely that difficulty of deglutition from disease of the trachea, thyroid gland, or carotid arteries will be overlooked. If the trachea is affected, dyspnœa will be a prominent symptom; if the thyroid gland, dyspnœa will also be associated with dysphagia, and the enlarged gland can be seen on the exterior. Disease of the vertebræ is not likely to cause obstruction in the œsophagus, for it would not press that organ against any other solid structure. The converse, however, is true: disease of the other structures causing difficulty of deglutition by pressing the œsophagus against the vertebræ. Within the thorax disease of the mediastinal glands and aneurism of the arch, or the descending portion of the aorta, enlarged left auricle or pericardial effusion, and disease of the left bronchus might cause constriction of the œsophagus. The mediastinal glands are enlarged from tuberculosis, carcinoma, or syphilitic disease. The occurrence of physical signs of a mediastinal tumor, with a history of syphilis or the general symptoms of tuberculosis or carcinoma, would point to the occurrence of these affections. In aneurism of the aorta in its arch or the transverse portion, the physical signs and subjective symptoms of aneurism, accentuation of the aortic second sound, and the presence of atheroma, would lend color to the view that the obstruction is of this nature. In both of the instances just mentioned the obstruction rarely goes to the extent of preventing the passage of liquids. In enlargement of the left auricle and in pericardial effusion the degree of difficulty may amount simply to a sense of obstruction or pain about the point where food passes these structures. Association of an enlarged auricle of mitral stenosis or of pericardial effusion with the early physical signs render the diagnosis of the condition easy. It is particularly important, in considering difficulty of deglutition from external pressure, to remember that the œsophagus is in close relation with the bronchus on the left side at about the fourth dorsal vertebra—this is ten inches from the teeth—in case it is desirable to investigate the obstruction with a probe. Obstruction from aneurism of the descending portion of the arch of the

aorta is also located at the upper portion of the œsophagus, nine inches from the incisor teeth.

2. Difficulty of deglutition due to disease of the œsophagus occurs in acute inflammation, in chronic inflammation, and in stricture, which is always the result of traumatic inflammation, of syphilis, or of cancer.

Acute inflammation is recognized by severe pain on swallowing. There may be tenderness on pressure along the course of the pharynx, and a feeling of a node in the lower portion of the throat. The pain is aggravated by speaking. The pain may extend along the vertebral column to the cardiac end of the stomach, and is usually of a burning or raw character. When the inflammation is due to traumatism, as to the swallowing of acids or other caustics, the mouth and pharynx show the effects of the inflammation, and in addition there is agonizing, burning pain at the root of the neck and between the shoulders. The inflammation is usually attended by erosion of the mucous membrane, and hence not only frothy mucus of a glairy character is expectorated, but also blood and shreds of membrane. The effect of the corrosive poisoning on the general system is marked. There is great prostration. Because of the accompanying gastritis there is intense thirst. Acute inflammation of the œsophagus may end in ulceration or in complete cure. The traumatic inflammation is followed by chronic inflammation, which ultimately results in stricture.

Chronic inflammation is attended by pain in the act of swallowing; viscid mucus is expectorated, usually in large amounts. Liquids are swallowed readily, but solids with great difficulty.

Abscess of the Œsophagus. The acute inflammation may terminate in abscess. Usually an abscess develops slowly, attended with pain on swallowing, increased by movements of the neck. When the abscess is high up in the gullet it may be seen on the exterior of the neck. If situated outside of the œsophagus and secondary to disease of the vertebræ, it is slow and chronic in its course; fever and rigors attend its development.

Stricture of the Œsophagus due to the healing of ulcers following traumatic inflammation is recognized, first, by the gradual development of the symptom, by the painless nature of the obstruction in the large majority of the cases, and by the seat of the obstruction. It is readily found if the tube is passed, or the patient can localize the area in the upper portion of the œsophagus. The difficulty of deglutition continues over such a long period of time that the nutrition is but slowly interfered with, but gradual emaciation with coincident anæmia sometimes develop.

Carcinoma of the Œsophagus. In cancer of the œsophagus, dysphagia is the most prominent symptom. It comes on gradually. The patient expectorates a considerable quantity of frothy mucus, often of blood, and on careful examination cancerous tissue may be found. Pain is not generally very severe. Cough is usually present, due to pressure of the cancerous mass on the recurrent laryngneal or pneumogastric nerve. Sometimes the cancer appears behind, and ulcerates into the trachea or bronchus. When this complication takes place the cough is violent. Dyspnœa from pressure is likely to occur. In the course of

cancer perforation of the œsophagus into the air-passages may take place with the occurrence of abscess and gangrene, or with dyspnœa and the onset of aspiration pneumonia.

The difficulty of deglutition due to cancer must be distinguished from traumatic or syphilitic stricture and from spasmodic stricture and paralysis of the œsophagus. The history of the case aids in the recognition of the former, while in spasm or paralysis the passage of the tube would point to the condition of the œsophagus. Cancer usually occurs late in life and is attended with rapid emaciation. The complications which ensue are attended with fever and rapid prostration. The cancer may be distinguished from disease outside of the œsophagus by the condition of the stomach beyond the point of stricture. If there is cancer, atrophy is more likely to take place, the diminution in size being recognized by a tube or by inflating the stomach with air or fluids.

3. Stricture or difficulty of deglutition from foreign bodies is usually recognized with ease. In the first place there is present a history of the swallowing of a foreign material. Sudden pain succeeds the act, while there is great anxiety and distress, particularly if the body is a large hard mass. Not only is there difficulty in deglutition, but also dyspnœa. The latter is due to pressure, but aggravated by the nervous state. When the foreign body is small the dysphagia is moderate in degree and the reflex irritation slight. If it cannot be removed ulceration and abscess take place, the further course of which depends upon the seat of the obstructing material.

Dilatation of the Œsophagus. Primary dilatation of the œsophagus is an extremely rare affection. The chief symptom is the regurgitation of food which is neutral or alkaline, and which may be returned some time after the act of swallowing. The patient sometimes complains of a sensation of distention along the course of the œsophagus, with heat and burning. The odor of the breath is fœtid. If the œsophagus is not deflected a bougie can be passed throughout its course.

If the dilatation is secondary the amount of dysphagia depends upon the obstruction. Food, however, is not returned immediately. After remaining an indefinite time, not longer than two hours, it is regurgitated unchanged. Bougies of course do not pass. In sacculated dilatation, which usually takes place in the posterior wall near the pharynx, a bougie may sometimes pass, and at other times may be caught in the sac. The sac may be enlarged so as to retain a considerable amount of food, which is regurgitated some time after it is swallowed. The sacculated diverticulum from traction on the outside of the œsophagus may occur when there is glandular disease of the neck with adhesions to the œsophagus. Traction occurs with the formation of the diverticulum.

Functional Affections of the Œsophagus. They are quite as common as organic disease of the œsophagus. The functional affections are of longer duration and unattended by grave effects upon the general system. *Spasm* is one of the most frequent affections. It may be so intense as to lead to stricture of a temporary character. It usually occurs in women. The attack comes on suddenly during the act of swallowing food. The food is at once regurgitated. After the subsidence of the perturbation that attends the attack swallowing can be

accomplished if the act is done slowly. It usually occurs in hysteria. The patient may have had some slight accident in the performance of the ordinary acts of deglutition which gave her the idea that she could not swallow. In consequence the further acts are performed with trepidation, and from slight emotional disturbance at the table sudden spasm takes place. The repetition of such spasm once or twice would be followed by a long régime of treatment. Unfortunately attention to the act of swallowing always embarasses it, and the taking of a meal under unusual circumstances is sure to be attended by complete dysphagia. Sometimes the idea is conceived that certain forms of food alone cannot be swallowed. It is usually solid food that is thought to give the distress. Mitchell says that the dysphagia occurs early in cases of hysteria; unless relieved the manifestations are likely to be transferred to the stomach. I saw a female patient who, after an ordinary choking attack could not swallow food if it was partaken in the presence of strangers or after the slightest emotional disturbance or hurry. The spasm disappeared after treatment with bougies.

In *paralysis* difficulty of deglutition is the main symptom, the course of which depends upon the cause of the paralysis. The larynx is usually affected at the same time, so that laryngeal symptoms are present. Paralysis generally comes on very gradually. It may be due to cerebral hemorrhage and tumor, and occurs in general paralysis of the insane and in bulbar paralysis. The bougie passes easily and does not cause irritation. In paralysis there is no regurgitation of food.

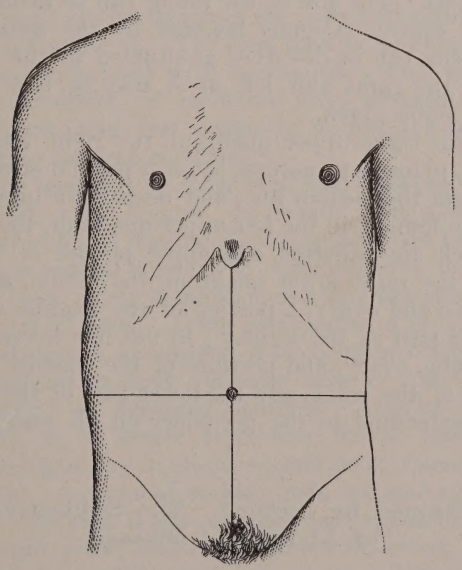
CHAPTER V.

DISEASES OF THE STOMACH, INTESTINES, AND PERITONEUM.

In the succeeding chapters diseases of the organs within the abdomen will be discussed. The subjective symptoms that attend diseases of the various organs call the attention of the observer to this portion of the trunk—the abdomen. Examination of the abdomen is made with a view to ascertain the special organ affected. It is proper, therefore, before a consideration of the diseases of each organ, to discuss the examination of the abdomen as a whole and the subjective symptoms referable to this region. It will be profitable to consider the topographical anatomy of the abdominal organs when the diseases of each are considered.

The abdomen is divided into various regions by vertical and transverse lines for the localization of organs or of disease. Unfortunately,

Fig. 76.



The quadrants of the abdomen.

the regions do not afford limitations for organs in health. Moreover, the regions are arbitrary, the boundaries differently constructed by various observers, and both are grasped with difficulty by the student. Simplicity should hold in these matters, and, moreover, a method of delimitation that is commonly used in the subdivision of other regions

should be adopted, to add ease of remembrance and uniformity of description. For these reasons, and because, as a teacher, I have seen the difficulties of students, the method of marking the surface prepared by Ballance appealed to me. This author includes the abdomen within a circle, with the umbilicus for its centre. The circle is divided into quadrants by diameters drawn at right angles, corresponding to the median and transverse umbilical lines. The portions to the right of the middle lines are the right upper and lower quadrants respectively; the portions to the left, the left upper and lower quadrants.

With the abdomen thus divided, the umbilicus and fixed bony structures in the periphery of the circle serve as points from which measurements are made to indicate the exact position of the structure the seat of which is to be recorded. The circle may be divided by other radii. To locate a tumor in the right lower quadrant, for instance, the umbilicus, pubic bone, and anterior spine of the ilium may be used as points from which to measure the distance. Measurements may also be made along radii extending from the umbilicus to fixed points. The following is a useful method: A tumor is situated in the right lower quadrant; the centre of the tumor is two inches below a point on the transverse umbilical line, three inches from the centre; it is also three inches to the right of a point on the median line, two inches from the umbilicus. The size of the tumor can be defined by measurements from its centre. Organs bisected by the median line, as the bladder and uterus, can be described as situated in the median line, as many inches to the right and left as it may be, and the number of inches from the pubis given.

Included in the right upper quadrant, the right lobe of the liver, the gall-bladder, pylorus, transverse colon, a portion of the pancreas, the pyloric orifice near the median line, and deeper, the upper half of the kidney would be found; in the left upper quadrant, the left lobe of the liver, the stomach, the pancreas, and upper portion of the kidney and the spleen; in the right lower quadrant, the cæcum, appendix vermiformis, right tube and ovary, a portion of the bladder and uterus, and above, the lower part of the kidney; in the left lower quandrant, the corresponding tube, ovary, and portions of the bladder and uterus, the sigmoid flexure of the colon, and the lower part of the kidney; about the centre and extending to the periphery on all sides, the small and large intestines.

The Data Obtained by Inquiry. The Subjective Symptoms of Abdominal Disease.

This class of symptoms will be discussed in the articles devoted to affections of the particular organs of the abdomen, because the symptoms are usually directly referred by the patient to the affected organs. They are local sensations of heat, fulness, or distention, of burning, of weight, or of undue motion. Local sensations of weight, fulness, or distention, are due to enlargements or to displacements of organs (liver, kidneys) or to tumors. Heat or burning is described in inflammatory

tumors, as pyosalpinx. It is often difficult for the sufferer to define the location of *pain* in the abdomen and describe its features. Moreover, the pain is frequently due to disease of the walls of the abdomen, a location which may cause confusion in the recognition of its true source. Pain must be investigated by an examination of each anatomical structure in relation to the part complained of as painful. The state of the function of each organ must be inquired into.

Pain due to Disease of the Structures of the Abdominal Walls. The skin, the nerves, the muscles and fascia, the connective tissue, may be the seat of the pain. If the skin is affected the pain is usually localized, not severe, and there are evidences of inflammation, as erythema, or ulcers, and there is superficial tenderness. Pain due to affections of the nerves is seen in herpes zoster and is recognized by the course of the pain and its attendant eruption. Neuralgias are recognized by the well-known points of tenderness, the intermittent character of the pain, and the association with anæmia; neuritis may be present, with the objective signs. A common cause of pain in the abdomen is due to disease of the vertebræ pressing upon the peripheral nerves at their emergence from the spinal column. It is situated in the median line, either below the ensiform cartilage or around the navel; it is an intermittent pain. Aneurism of the abdominal aorta with pressure and erosion causes the same character of pain. The muscles and fascia may be the seat of rheumatic inflammation, causing severe pain. The muscles may be tender. Movement always increases the pain, and sighing, laughing, or coughing may aggravate it. The pain may be so diffuse and severe as to lead it to be confounded with peritonitis. The presence of rheumatism in other muscles, of moderate fever without gastro-intestinal disturbance, of uric acid and urates in excess due to the rheumatic diathesis, points to the true condition.

The *seat* of the pain will be considered in discussing special organs or diseases of the individual organs. In general it may be said the seat of the pain is a fair index of disease of some structure in the part indicated. When the pain is general it points to rheumatism or to peritonitis.

Character of Pain. Pain in the abdomen may be acute or may continue over a long period of time. Acute pain points to inflammation, to perforation, to gastralgia, to enteralgia, or to occlusion of channels, of which the abdomen contains so many; chronic pain, to ulcer, to chronic processes, or to gastric or intestinal neurosis. Attacks of pain may be sudden in onset, or, in severe type, may be the result of a gradual increase of pain, beginning in slight sensations of discomfort.

Mode of Onset. Attacks of sudden pain are spoken of as *colic*; the onset is sudden; the pain is paroxysmal; each spasm of pain is attended by vomiting, rapid pulse, cold extremities, cold sweat, and more or less collapse, except in lead colic. Such pain is seen in intestinal colic, hepatic colic, renal colic, uterine and vesical colic.

Sudden pain occurs in perforation of some one of the hollow viscera, its seat being ascertained by the history of the disease prevailing at the time, the location of the disease, and the character of the symptoms attending the pain. Thus perforations of gastric ulcer may have occurred in the course of the disease, the symptoms of which were

previously present. The seat of the pain would point to its source, and the occurrence of vomiting aid in detecting its origin.

The Data Obtained by Observation. The Objective Symptoms.

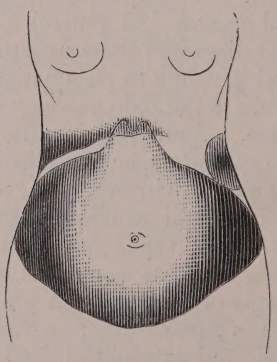
The examination to determine objective symptoms of disease within the abdomen is made by the usual methods. Changes in the appearance of the abdomen are caused by disease of structures adjacent to the abdomen, or remote from it, as the lungs or heart, or the brain. Disease or paralysis of the diaphragm alters the movements of the abdomen in respiration and the appearance of the upper half. Fluctuating changes in size occur in hysteria and gastric neurasthenia and permanent change in tuberculous meningitis. The objective signs are observed by the usual methods.

Inspection. In general inspection of the abdomen, attention should be directed, first, to the size and shape; second, to the color and to the presence of normal or abnormal markings; third, to pulsations and unusual movements of some of the viscera, to the condition of the abdominal walls and the appearance of the veins. Increase in size may be *general* or *local*.

The abdomen differs very much in size in different persons, depending not only upon the thickness of the fat in the abdominal walls and omentum, but upon the calibre of the intestines themselves, which are apt to be much distended in those accustomed to eat large meals. In general, the belly is more protuberant in infants and children than in adults.

General Enlargement of the Abdomen. This occurs in obesity, and it is often difficult to tell whether the excessive deposit of

Fig. 77.



The shading indicates the position of the percussion dulness in a case of ascites, while the patient is lying on the back, the fluid falling to the low levels in the flanks, and the umbilical region remaining clear. (Finlayson.)

fat in the abdominal walls and omentum accounts for the whole enlargement or only serves to mask the presence of a tumor. Enlargement of the belly from obesity is only a part, though frequently the most pronounced evidence of obesity, whereas, in enlargements of the abdo-

men from other causes than flatulency, such as tumors and ascites, there is usually a marked contrast between the size of the abdomen and that of the rest of the body.

In enlargement from *ascites*, when the patient is lying upon his back the front of the abdomen is flattened, while the flanks bulge. If he turns upon his side, the flank which is uppermost becomes hollowed out and the front of the belly is prominent. This is the appearance in moderately large effusions which have existed long enough to stretch the lateral abdominal muscles. When the effusion is enormous, all parts of the belly are distended, and the appearance of the abdomen is then barrel-shaped; and no change of shape occurs upon change of posture. (Fig. 77.)

Enlargement from *accumulation of gas* within the bowels is general, and may attain a very high degree, giving the abdomen a uniform arched appearance resembling a barrel. The diaphragm may be pressed upward so far as to interfere seriously with respiration and heart action. Moderate degrees of distention from gas in the intestines may be the result of eating certain articles of food, such as turnips or beans. Excessive accumulations are met with in typhoid fever; peritonitis, operative and non-operative; and in stenosis of the colon or rectum from any cause. They are also common in hysteria.

In the last month or two of *pregnancy* enlargement of the abdomen is general, especially in a woman who has previously borne children.

General enlargement of the abdomen may be due also to cancer of the peritoneum, to hydatid cyst, and to cancer of the bowel. It has been observed in children in dilatation of the colon. The abdomen was uniformly enlarged in Hughes' case and in Osler's cases. Coils of the intestine, with waves of peristalsis were seen through the thin abdominal walls. Formad's case occurred in an adult. The distention was enormous. Constipation attended all these cases.

Other causes of abdominal enlargement are diseases of the *liver* and *gall-bladder*. When these are considerably enlarged a local swelling may be detected in the right upper quadrant; but when they attain very large dimensions, as happens not infrequently in cancer, amyloid disease, and hydatid liver, inspection may be able to detect only general enlargement, with small prominences corresponding with cancerous nodules or small cysts.

Splenic enlargements, which attain the greatest size, are from leukæmia or chronic malarial poisoning, and are usually visible only as general enlargements of the belly. There may, however, be greater prominence over the lower left ribs and in the left upper quadrant posteriorly.

In diseases of the *kidney* producing great enlargement there is usually visible a prominence in the lateral and lumbar region of the kidney involved, unless there is considerable emaciation, but anteriorly the enlargement, if any be visible, usually appears to be general.

Enlargements of the abdomen which begin in the lower quadrants are usually of pelvic origin. The most common are those due to *pregnancy*, *cysts* of the ovary or parovarium, *fibroids* and *fibro-cysts* of the uterus, and abscesses or effusions (chronic peritonitis). A greatly *distended bladder* may cause confusion; it is a good rule to be sure that the bladder is empty, by having a catheter passed, before proceeding further

with the examination. Intestinal peristalsis is observed in constriction of the bowels. The motion of the intestine above the seat of stricture is wave-like or worm-like, and the bowel itself dilated.

Local Enlargement, or Tumors of the Abdomen. In the space below the xiphoid cartilage and between the ribs (epigastrium), local enlargements may be due to a distended or dilated stomach, or to a tumor of the pylorus, which is almost always cancerous. But enlargement in this region is sometimes due to cysts, sclerosis or cancer of the *pancreas*, to *aneurisms*, to cancer of the large intestine or the left lobe of the liver. It is in this region or to the left of the median line and nearer the umbilicus that the effusions into the lesser peritoneal cavity are found.

A rigid rectus muscle is capable of simulating a tumor, and in hysterical subjects when associated with tympanites has received the name, phantom tumor. Such swellings are less constant in their shape and character than genuine tumors, and while dull on percussion, may be detected to be more superficial; they sometimes disappear under friction, and certainly under full anæsthesia; nervous symptoms are present and decided effect upon the health absent.

Enlargements in the *right upper quadrant* (right hypochondrium) are most frequently due to diseases of the *liver* (which see), and to affections of the gall-bladder. But less frequently a much enlarged kidney or a hydronephrosis causes swelling in this region. The differential diagnosis is made by the history of the case and by noting the direction in which the tumor has grown, by examination of the urine, and by the relation which the ascending colon bears to the tumor; kidney tumors carry it in front of them as they grow; hence their dulness is obscured by the superficial tympany of the colon.

Enlargement in the *right lower quadrant* (right iliac region) is most frequently due to affections of the cæcum and appendix, to tumors of the ovary, and to pelvic abscesses.

The diseases of the *cæcum* and *appendix* causing enlargement in the right iliac fossa are fæcal accumulation, typhlitis, fæcal abscess, perityphlitic abscess, and stricture of the ileo-cæcal valve.

The diseases of the *ovaries* and *tubes* causing enlargement in this region are ovarian tumors, cysts of the broad ligament, pelvic abscess (usually tubal in origin), and extra-uterine pregnancy.

Other affections which need to be considered are *tubercular* peritonitis, acute and chronic, and rare instances of disease of the kidneys or spleen with considerable enlargement.

Enlargement in the *left upper quadrant* (left hypochondriac region) is due to dilatation or carcinoma of the stomach; enlargement of the spleen, movable kidney, or tumors of the kidneys, and effusion in the lesser peritoneal cavity. Enlargement in the *left lower quadrant* (left iliac region) is due to tumors (cancerous) of the sigmoid flexure and to the tumor due to volvulus, and causes of enlargement of the right side which are possible on the left.

Enlargement about the *centre of the abdomen* (umbilical region) may be due to umbilical hernia, to a floating kidney, spleen, or liver, or to tubercular disease of the omentum or mesenteric glands. This region

is frequently enlarged in conjunction with a more prominent swelling extending from the sternum in cancer of the stomach, from the ribs on the right in cancer of the liver or gall-bladder, or other disease of these viscera, the ribs on the left, in effusions into the lesser peritoneal cavity, disease of the pancreas or the spleen. Undue projection of the vertebræ must not be mistaken for tumors.

Enlargement above the pubis (hypogastric region) is due most frequently to enlargement of the uterus, from pregnancy, fibroid tumors, or fibro-cysts, or to distention of the bladder.

Enlargement in the lateral regions and behind (*lumbar region*) occurs in malignant tumors of the kidney, in hydro- and pyo-nephrosis, in peri-nephritic abscess, and in renal cysts of large size. It may also, in the left side, be due to perigastric sub-diaphragmatic abscess, and to enlargement and displacement of the spleen. On the right side the cause may be enlargement of the liver or a hydatid cyst.

Diminution in Size. The abdomen is diminished in size in wasting diseases, or such as result in insufficient food being taken. Among this class come cancer of the œsophagus and stomach, chronic lead-poisoning, anorexia nervosa, and chronic diarrhœa and tuberculosis of childhood. In tubercular meningitis in children there is retraction of the abdomen in the second stage. The wasting of the subcutaneous and the omental fat, and atrophy of the abdominal organs, cause the abdomen to be concave or *scaphoid*.

The Shape. In general enlargements the shape is uniform. In large accumulations of fat in women with relaxed abdominal walls the abdomen may be pendulous. In ascites the tissue over the umbilicus may protrude, changing the uniform appearance. In local enlargements the surface is often irregular, corresponding to the seat of the enlargement. The shape changes in hysterical distention. In enlargement due to wasting disease of the viscera, as cancer of the retro-peritoneal glands, the abdomen retracts in the later stage of the disease, causing undue prominence of the viscera affected by carcinoma.

The Color. The abdomen, in general, partakes of the hue of the skin of the body. It is darker around the umbilicus. In Addison's disease a distinct areola often forms. The median line, from the umbilicus to the pubis, darkens in pregnancy—the "brown line." It is sometimes seen in men. The skin of the abdomen is the seat of specific eruptions, as in typhoid fever, and of sudamina. The walls may be pale and glistening in œdema.

Markings. In first pregnancies and great ascites, less frequently in obesity and tumors, *striæ* are produced in the parts of the skin where the tension has been greatest. In pregnancy they form sinuous lines upon the lower lateral portions of the abdominal wall and upon the upper inner portions of the thighs. When first developed they are reddish, but subsequently become, by a process of fading, more glistening and white than the rest of the skin. They are also known as "water lines," or *lineæ albicantes*.

The Movements. (See the Lungs—*Dyspnœa.*) The upper zone participates in respiratory movements, especially in males. In enlargement of the abdomen and in upper abdominal tumors, the movement is

restricted. Abdominal pulsations are observed. The liver may be the seat of pulsation. The region below the sternum (epigastrium) is a common seat, but pulsation may occur anywhere in the course of the aorta. (See Epigastric Pulsation, p. 368.)

Peristaltic movement may be seen through the abdominal walls. It may occupy the large or the small intestine. If the large intestine, the waves are confined to the seat of this canal; if the small intestine, to the region around the umbilicus. It is always due to obstruction of the lumen of the bowels. It is also seen in dilatation of the stomach. Pulsation of the liver may occur. (See Dilatation of Heart.)

The Veins. Enlargement of the *superficial veins* is a common accompaniment of cirrhosis of the liver, and stasis of the portal circulation, as well as of any cause which obstructs the free circulation in the inferior vena cava. Occasionally a varicose condition of the veins about the umbilicus is seen (*caput Medusæ*).

General Palpation and Percussion of the Abdomen. Palpation and percussion in diseases of the abdomen may be discussed together. Generally the best position is the recumbent one, because it admits of examination without too great exposure, and because in that position the abdominal muscles are partly relaxed. When the muscles need to be still further relaxed the patient should lie upon the back with the head and thorax partly elevated and the knees drawn up. The examining hand should be warm, as the application of a cold hand throws the abdominal muscles into involuntary contraction. In certain obscure tumors much can be learned by having the patient rest on the hands and knees, or assume a knee-chest position. By this means we can determine if the pulsation is due to aneurism or to a tumor. The latter falls away from the vessels, and lessens pulsation thereby in the position just mentioned. A tumor surrounded by coils of intestine may be more palpable.

Moreover, by grasping the abdominal walls between the thumb and fingers their thickness and the relative proportion of *fat* can be estimated. So, too, the presence or absence of *œdema* of the skin can be readily detected. This œdema is general, but especially marked in the lateral and posterior portions of the abdomen. Relaxed abdominal walls occur after dropsy and pregnancy. Redundant skin remains in folds when pinched up. This is particularly so in abdominal cancer.

When it is desired to explore deeply the patient should be instructed to breathe with the mouth open, and the examining hand pressed firmly in during respiration, and held there, if need be, during several long breaths. The same procedure is adopted when we desire to get the percussion note of a body lying deep in the abdomen: the finger is pressed firmly and deeply in, and then percussed. In this way any superficial resonance due to overlying intestine is eliminated.

When palpating to determine the lower edge of the liver or spleen, the palmar surface of the fingers is pressed into the abdomen at different levels from below upward until the edge is felt. The edge of the right lobe of the liver in its normal position extends to the margin of the ribs. It may be detected by pressing the fingers in as described and having the patient take a long breath.

By palpation, the facts derived by inspection are confirmed; the character of the abdominal walls and of swellings determined; the precise location of pain ascertained; the condition at the hernial rings, and the movability of tumors inquired into. The condition of the integument should first be determined. Passing the hand gently over it is sufficient to decide whether it is normally smooth and elastic, or harsh and dry. Any marked unevenness, such as are produced by umbilical and inguinal herniæ, striæ, or by large tumors of the pylorus, or cancerous nodules and hydatid cysts of the liver, can readily be detected. The degree of tension of the abdominal walls is easily appreciated. It is increased, of course, in all forms of great enlargement, but not equally; and some persons are so sensitive to touch that any attempt at palpation throws the abdominal muscles into such rigid contraction that examination is impossible. Rigidity of the abdominal walls may be the only sign of acute peritonitis. It is common in local peritonitis. The recti muscles contract quickly on hurried palpation. Local contractions point to inflammation underneath. In tuberculous peritonitis we see distention with board-like rigidity or preternatural hardness. The term *carreau* is applied by the French to this condition. *Peritoneal friction* may be detected most frequently over the liver and in chronic peritonitis.

Palpation and Percussion of the Lower Quadrants. On the right side, the groups of affections connected with the cæcum and appendix, the uterine appendages, and the peritoneum, which cause enlargement in this region, have been mentioned already under local inspection of the abdomen. Palpation and percussion, however, are the methods which afford exact information of their physical characteristics and, with the clinical history, enable us to differentiate one from the other.

Diseases of the Appendix and Cœcum. The information supplied by palpation and percussion in perforation of the appendix will depend upon the rapidity with which perforation has supervened and upon the stage at which the examination is made.

Speaking generally, following the sudden onset of pain in the right iliac fossa in a person previously in good health, tenderness on palpation in this region is felt. This tenderness is first localized, but may spread with great rapidity over the whole abdomen. Subsequently, the tension in the part is increased, the percussion resonance impaired, and there may be a gurgling sound on pressure with the hand. Examination with the finger in the rectum may discover the presence of a tense, swollen appendix, or of a tumor in the pelvis.

But the disease may be fulminating in character, perforation being followed by the rapid development of peritonitis, with collapse, so that when the patient is seen there will be no more tenderness over one part of the abdomen than another.

Again, the appendix may be subject to repeated attacks of inflammation without perforation, but with the development of local peritonitis. There is increased thickening in the region of the cæcum, tenderness, diminished resonance, and increased resistance to the percussed finger. Sometimes an enlarged and hardened appendix can be made out by palpation, both during an attack and in the intervals.

In still other cases, of slower development, a distinct perityphlitic abscess develops. In addition to local pain and tenderness, a swelling appears above Poupart's ligament. The skin over it becomes brawny and pits on pressure with the finger-tips. The tumor is dull on percussion, and on palpation obscure deep-seated fluctuation can be obtained. A fluctuating tumor can also be made out by rectal examination with the finger.

In *fæcal impaction of the cæcum* a tumor forms, following the course of the cæcum and being directed upward from Poupart's ligament. It is usually oblong and rounded, and may be uneven or lumpy on closer palpation; it is not tender unless the cæcum itself becomes inflamed. It has a doughy consistency.

The diagnosis is made by the situation and character of the tumor, the absence of pain, tenderness, and constitutional symptoms, and by its disappearance under the influence of purgatives.

In *typhlitis* a sausage-shaped tumor is found lying above Poupart's ligament and running upward from it. It is frequently the result of fæcal impaction of the cæcum. The tumor is tense, tender, and painful, dull on percussion, the dulness being sharply limited by the boundaries of the cæcum.

In *intussusception* a tumor is often detected in the right lower quadrant or to the right of the navel. It is generally distinct, of the shape of the bowel, not very tender, and harder than the tumor of appendicular inflammation. The diagnosis from the latter is made by the difference in the character of the tumor, by the pain being colicky and recurring in paroxysms, by vomiting and constipation being more marked, and by the passage of blood and mucus from the bowel. The last named and the tumor, with a constant desire to defæcate, are the most characteristic symptoms. A tumor may be detected within the rectum by digital exploration, if the intussusception is low down. There may be distinct hemorrhage, or the passage of the invaginated portion of the bowel per rectum. Intussusception is the most frequent cause of intestinal obstruction in infants and young children. It occurs nearly twice as often in males as in females. Stercoraceous vomiting is not so common as in other forms of acute obstruction of the bowel. The affection is of short duration, ending in recovery or death, usually within a week. Exceptionally, life may be prolonged for a much longer time.

In *pelvic abscess* a swelling sometimes makes its appearance on the right side above Poupart's ligament. It is, perhaps, situated more toward the median line than perityphlitic abscess, and it is less defined than the tumor of typhlitis; but the diagnosis from these affections must be made by the history, which is usually that of an antecedent salpingitis, or of previous abortion or miscarriage. Vaginal examination discovers that palpation of the uterus causes pain; that the uterus is fixed in position, instead of being freely movable; and that the pelvis is blocked up by an exudate on the affected side.

In *pelvic hæmatocele* a tumor may form and be discovered in the lower half of one of the lower quadrants. It is distinguished from appendicitis, perityphlitic abscess, and pelvic abscess by the absence of fever and constitutional signs of suppuration; from perityphlitic

and pelvic abscess by its sudden onset, probably at a menstrual period; by the less degree of tenderness; by the anæmia and collapse which have followed its appearance. It is almost invariably the result of a ruptured *extra-uterine pregnancy.* From pelvic abscess it is distinguished by its occurrence in a woman without antecedent tubal or uterine disease, and by the less degree of tenderness of the uterus and relative absence of fixation.

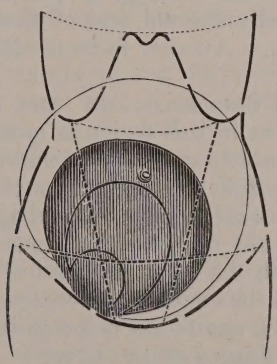
In *stricture of the ileo-cæcal valve* due to cancer there is frequently a tumor in the right lower quadrant between the umbilicus and anterior superior process of the ilium, or the latter and the ribs. The diagnosis is made by noting the fact that the tumor has developed gradually, that the patient has suffered with colicky pain, vomiting and constipation, possibly preceded by diarrhœa, and that peristaltic movements of the intestines can readily be seen through the abdominal walls. The abdomen at the site of the tumor is somewhat distended. The tumor itself is irregular and tender, and is dull on percussion.

The disease is very rare, and is said by Fenwick to be more common in women from twenty to forty years of age.

Fæcal abscess, arising from ulceration of the colon, may be suspected, according to Fenwick, when there is a localized abdominal swelling, immovable in respiration or by a moderate amount of pressure with the fingers, the size and shape being altered when diarrhœa occurs, and when percussion over the tumor gives a tympanitic or a more forcible stroke, a dull sound, or when an emphysematous sensation is communicated to the fingers.

In *tumors of the right ovary* there is at first a gradual enlargement in the right groin, unaccompanied by pain, fever, or impairment of health

FIG. 78.



Position of an *ovarian tumor* of the right side, in various stages of enlargement. The shading indicates the *percussion dulness in ovarian dropsy of moderate extent:* the umbilical region is dull, from the presence of fluid, and the flanks remain clear. The outer circle shows a further extent to which the dulness may reach in ovarian dropsy. (BRIGHT.)

until the tumor has attained considerable size. They are usually cystic, and fluctuation can be obtained. The tumor is dull, and by bimanual examination with the fingers of one hand in the vagina the tumor can be traced into the broad ligament and the displacement of the uterus

which it occasions made out. The cystic ovarian tumors grow from the starting-point in the direction of an axis diagonally toward the median line. There is dulness in front of the abdomen, a clear percussion note or tympany in the flanks. (Fig. 78.)

In the Left Lower Quadrant. Enlargements in this region are due most frequently in women to *ovarian tumors, pelvic abscess, pelvic hæmatocele, and fibroids of the uterus,* the diagnostic points of which have been referred to already under palpation and percussion of the *right* iliac region. In addition to the affections named, enlargements are occasionally met with from fæcal accumulations in the flexure of the colon, *cancer of the descending colon, tubercular peritonitis,* and enlargements or displacements of the *spleen* and *kidney* (which see). *Fæcal abscess* also may occur here, and the tumor of *intussusception* may be detected on the left side.

Palpation and Percussion above the Pubis. Enlargements in this region may be due to *fibroid tumors of the womb.* They occur most frequently in sterile women, and are accompanied usually by hemorrhage. Bimanual examination of the uterus will be able to detect an unevenness of surface of the womb if the tumor is external, and passage of the sound will detect any growth projecting into the cavity of the womb.

The enlargement may be due to *a distended bladder.* It is a good rule always to be sure that this viscus is empty before beginning an examination.

In *acute tubercular peritonitis* a swelling may develop in this region. It appears gradually, is diffused and free from tenderness, but is preceded by pain and fever. There is no palpable tumor, but the percussion note is dull and the tension is increased. Moreover, the level of dulness is apt to vary with change of posture of the patient. The general health is markedly affected, loss of flesh is rapid, and diarrhœa and sweats are common. Another focus of disease may be discovered in the lungs.

Palpation and Percussion of the Region below the Sternum. Enlargement in this region is most frequently due to affections of the *stomach* (which see). It is not uncommon, however, to find here a cancerous nodule projecting from the surface of the *liver,* or an hydatid cyst of the same organ. The diagnosis must be made by determining with the aid of palpation and percussion whether the tumor is continuous with the liver, the effect of respiration upon it, and its apparent depth from the surface, tenderness, fluctuation, etc., and by a study of the subjective symptoms pointing to disease of the stomach or liver. (See under Diseases of the Liver.)

Much more rarely enlargement here may be from tumor of the *pancreas,* which may be from cyst, abscess, or from cancer. According to the studies of Fitz, the former is marked by deep-seated colicky pain occurring in paroxysms, by discharges from the bowels of matter resembling saliva, by the detection of fat in the stools and sugar in the urine, by salivation, and by the occurrence of jaundice.

Cancer of the pancreas is to be recognized by the detection of a painful tumor in the epigastrium. The pain is not aggravated by the

taking of food, but is said to be by the erect posture. The bowels are constipated, and the stools may or may not be fatty. Emaciation is progressive, as in all cancerous affections, and in the latter stages there may be occasional vomiting and persistent jaundice.

Palpation and Percussion of the Upper Left Quadrant. Enlargement in this region is generally due to disease of the *spleen* (which see).

It may be due to *fæcal accumulation* in the left transverse and descending colon. This condition is recognized by the painlessness and doughy consistence of the tumor, and by careful inquiry as to the condition of the bowels. Constipation will, of course, exist, but both patient and physician may be misled by apparent diarrhœa, or even dysentery; there will be fluid or semifluid dejections mingled with scybala, and sometimes mucus and blood.

An interesting cause of swelling in this region, and in the lumbar region, is *perigastric*, or *subdiaphragmatic abscess*, a collection of pus walled in by the stomach, spleen, diaphragm, colon, and the abdominal walls.

The most common cause is the irritation of a gastric ulcer which has nearly or quite perforated, and has formed adhesions with surrounding viscera. This was the cause in forty-one out of fifty-two cases analyzed by Fenwick, while in six it was associated with cancer and in four with abscess commencing externally. Pain in the epigastrium or abdomen was the chief subject of complaint, and in most of the cases there was dyspepsia, sometimes with vomiting. It is singular that hæmatemesis was mentioned in only two cases. Fenwick thinks that in every case of perigastric abscess, except in persons affected with phthisis, cancer, or some other chronic exhausting malady, the first formation of the abscess will be accompanied by either collapse and signs of general peritonitis, or by sudden and severe pain in the epigastrium, attended with indications of local peritonitis.

Fever is a prominent symptom, but physical signs are absent. A tumor, according to the same author, is rarely distinguishable except when the cause is cancer. It is dull, but afterward tympanitic on percussion, and not movable on inspiration or external pressure. The tension of the abdominal muscles prevents successful palpation. There may be arching outward of the ribs. The displacement of surrounding viscera will depend upon the size of the abscess and the extent of adhesions. But the diaphragm is pushed upward, and dulness may extend as high up as the angle of the scapula. In this case a pleural effusion is simulated. Breathing is embarrassed by the upward pressure of the lung and heart. Sometimes when gas is formed in connection with the abscess amphoric sounds on auscultation and percussion are heard both in the abdomen and over the thorax. To this condition the name *pyo-pneumothorax subphrenicus* has been applied. The abdomen then becomes tense, tender, prominent, and tympanitic on percussion.

Palpation and Percussion of the Loins. Enlargements in these regions are due most frequently to affections of the *kidney* (which see). They may, however, be due to enlargement or displacement of the

spleen and *liver* (which see), or to tumors of the retro-peritoneal glands. On the left side the possibility of *perigastric abscess* must be borne in mind, as sometimes the dulness and increased tension of the tumor extend as far down as the lumbar region.

Palpation and Percussion about the Centre of the Abdomen. *Umbilical hernia, cancers of the stomach and liver, hydatid cysts of the liver,* and *tumors of the gall-bladder,* together with *floating kidney, spleen,* and *liver,* all at times cause tumors which may be felt in this region. They must be distinguished from each other by methods already referred to under the organs named. The general principle upon which to proceed is to endeavor by palpation and percussion to discover the organ to which the tumor belongs. To this end also careful inquiry should be made as to the time the tumor has been known to exist; its effect upon the general health, if any; its effect upon the function of the possible organs affected, and particularly as to the presence or absence of vomiting, constipation, diarrhœa, or jaundice.

Tumor in the region about the umbilicus may be from *tubercular disease of the mesenteric glands* (*tabes mensenterica*). It occurs nearly always in children, and presents the physical signs and symptoms of tubercular peritonitis, with the addition that enlarged mesenteric glands may sometimes be felt. Children grow pale and anæmic, waste away, have apparently causeless diarrhœa, the passages being foul and the food undigested. The abdomen is large, but appears larger when compared with the emaciated body. It is tender, its walls thickened, and less elastic than normal. Signs of tubercular disease in other organs may be detected.

Facts gathered in this way, carefully analyzed, and then studied with reference to the physical properties of the tumor (hard or soft, fluctuating, doughy, or not), will generally suffice for a probable diagnosis. A positive diagnosis often cannot be made at the first examination, and sometimes is possible only after watching the progress of the case for a considerable time.

Diseases of the Stomach.

The stomach is a canal in which the food is detained for the purpose of solution. Its walls are made up of mucous membrane, muscle and peritoneum. It is richly supplied with bloodvessels. Because of its great functional activity it has an abundant nerve supply. It is, moreover, surrounded by rich plexuses of sympathetic nerves, through the influence of which and its special nerve, the pneumogastric, it is in intimate relation with every organ of the body.

The Symptomatology. The local symptoms of disease of the stomach are dependent upon: (1) The morbid process which affects it; (2) the effect of the process upon the anatomical structure of the organ (atrophy, dilatation, tumor) whereby the size is affected; (3) the effect upon its function.

The symptoms due (1) to the morbid process are not different from the symptoms of similar morbid processes, save that they are modified by

the function of the organ or its special anatomy, a canal. Hence congestions are attended by discharge of mucus; inflammations are attended by pain and by a flow of mucus and pus; ulcers by pain and the accidents of ulceration (hemorrhage); malignant disease by pain and swelling (tumor), and its accidents, hemorrhage and obstruction; while to each process belong the general phenomena which attend it. But the stomach is highly sensitive and resents the intrusion of a process or of that which (1) causes or (2) irritates the process. Expression of this resentment is shown in the occurrence of hyperæsthetic symptoms (see the Neuroses), as of pain, in the abolition or derangement of function, and in the occurrence of the great pathological reflex act of the stomach—vomiting. It will be seen later that this is a symptom of every local morbid process of the organ, either directly because of the process or on account of the cause of the process, both of which are operative in inflammation due to any irritant; or indirectly because the process has set up undue sensitiveness. In the latter instance material, as food which the stomach is accustomed to receive, becomes an irritant. Abnormal material from morbid processes acts as an irritant, as mucus, pus, or blood.

The morbid processes modify the anatomical structure and lead to other morbid conditions, as we see when dilatation succeeds inflammation or obstruction of the orifices. Now the symptoms of the secondary conditions are the symptoms of such elsewhere—in atrophy, diminution in size; in dilatation, increase in size, with retention and fermentation, and finally discharge of contents by vomiting.

Functional Symptoms. Any local disease of the stomach must influence its function; therefore, conversely, functional symptoms must be present in all local diseases. The functions of the stomach are to digest and to absorb the products of digestion. The former function is motor and chemical, the completeness of which depends upon mixture of the food with, and solution in, the gastric juice. The symptoms, therefore, must be due to changes (1) in the motor, (2) in the secretory, and (3) in the absorptive function of the organ.

Central and Reflex Influences. In the consideration of the symptomatology of gastric diseases the anatomical relations through the influence of its vascular and nervous connection must be considered. The student is sufficiently familiar with physiology and pathology to know that each organ has a representative in the central nerve mass, the brain, and that disease in one organ will influence the function and create morbid symptoms in another which may happen to be related to it through intimate nervous connections.

The central representative or centre is influential in degree in accordance with the power and activity of its peripheral adjunct. It is, moreover, regulated by higher centres, the psychical, and it in turn modifies them. It influences or modifies lower centres, (1) functional, (2) vasomotor, (3) motor, or (4) sensory. The result of this mechanism is: 1. That functional alteration or organic disease of (*a*) the gastric centre, or (*b*) of centres of higher control, or (*c*) of the nerve that connects centre and organ, pneumogastric, produces gastric symptoms. 2. That gastric diseases produce symptoms in other organs, as palpitation of the

heart (reflex). 3. That disease of other organs produces gastric symptoms or disease, as the vomiting of pregnancy, or renal calculus, or disease of the testicle, or the gastritis of nephritis. Thus, vomiting is caused by emotion (high centre) influencing the (lower) pneumogastric centre; by a tumor pressing on or destroying the pneumogastric centre; or by a tumor pressing on the pneumogastric nerve, as aneurism. I have taken the simplest illustration. When we come to the study of gastric neuroses the extraordinary influences of the nervous mechanism will be appreciated; or when hysteria is studied, the mechanism of its extreme gastric symptoms will be recognized in a measure. To continue with vomiting: when its mechanism and clinical course is studied it will be found to be due to affections of the blood, the poisons of which irritate cerebral centres or nerve plexuses in the stomach.

But gastric diseases also arise because of their vascular connection. Thus, in heart disease with venous congestion the gastric veins become the seat of congestion with the production of gastric catarrh. Or hepatic disease will cause portal congestion and gastric catarrh.

It is observed, therefore, in unravelling the symptomatology of gastric disease, we must first note (*A*) the subjective symptoms due to (1) possible morbid processes, (2) to alterations of function, (3) to alterations of size (sense of fulness, etc.). (*B*) The objective symptoms due to (1) morbid processes, (2) to alterations of function, (3) to alterations of size.

Now one of the objective expressions of the *morbid process* or of altered function is seen in changes in the character of the contents of the stomach. The contents are obtained for examination when discharged from the stomach (vomit) or when removed artificially (washings). Both fluids are studied by inspection, including microscopical examination, by smelling to note the odor, and by chemical and bacteriological examination. Alteration of function is also seen in alteration of digestion, and is estimated by chemical and physiological methods. The activity of the digestion must be determined by ascertaining the duration of digestion and its degree of completeness, which depends upon three factors: (1) The motor power; (2) the absorptive power; (3) the digestive power of the secretions the activity of which is investigated.

To secure objective data, therefore, the following is necessary:

I. Physical examination to determine tenderness and the size and position of the stomach.

II. Examination of gastric contents:

1. Character of secretion.
2. Amount of secretion. (HCl.)
3. Determination of the power of digestion of—
   a. Albumin.
   b. Milk.
   c. Starch and sugar.
4. Determination of the motor power.
5. Determination of the absorptive power.
6. Examination of the vomitus.

Further Examination. In addition to the examination of the stomach in order to judge correctly of the nature of gastric lesions as indicated above, we must ascertain (1) whether the gastric symptoms are dependent upon disease of other organs, particularly the eye, nose, and genitalia, the heart and kindeys, by an examination of each organ, and (2) whether other symptoms are created by gastric disease.

Toxic Symptoms. There is one class of symptoms that arise in gastric disease that are worthy of a few words. They are nervous symptoms due to the absorption of ptomaïnes or imperfect products of assimilation, on account of which, if absorption takes place suddenly and in large amounts, coma and convulsions occur; or, if chronic, hypochondriasis, melancholia, mental depression, with vasomotor phenomena of various kinds, arise.

Diagnosis from disease of contiguous organs functionally related. The student will soon learn that diseases of the stomach which are functional in character cannot be differentiated with ease from diseases in other organs functionally related. He will find that to draw hard-and-fast lines between gastric and intestinal indigestion, or between so-called disordered gastric and hepatic function, is impossible. Organs which are closely related in physiological function, and which have nerve and blood supply in common, cannot be differentiated when disordered function is considered. Hence indigestion and biliousness, or simple acute gastritis and duodenitis, are beyond the pale of close discrimination. In fact, the symptoms of each blend, in a manner.

The Data Obtained by Observation. The Objective Symptoms.

Physical Examination of the Stomach. Inspection. Direct inspection of the stomach rarely affords much positive information. When there is much loss of abdominal fat and the stomach is well distended, its outlines can sometimes be traced with the eye. The best position is behind and above the patient's head while he is lying down. If the lower curvature can be traced considerably below the navel the stomach is almost certainly dilated, and if, at the same time there is a prominent swelling in the pyloric region, accompanied by progressive loss of weight and cachexia, the dilatation is probably due to cancer of the pylorus.

Peristaltic waves may be seen with the naked eye, or brought into view by the use of the ether spray or faradism. When the pylorus is obstructed anti-peristaltic waves may also be seen. The waves of muscular contraction begin at the cardiac end or fundus, and extend to the pylorus; hence they begin under the ribs of the left side and extend downward toward the right lower quadrant. They vary in extent with the amount of dilatation.

An endoscope has been adapted for inspection of the stomach; but such an instrument necessarily can be in the hands of but few, and it would be difficult to persuade American patients to permit its use.

Distention of the stomach with carbonic oxide or air frequently brings the outlines of tumors of the pylorus plainly into view, while at the same time any tumor lying behind the stomach becomes less distinct, and

false tumors due to spasm of the gastric muscular coat vanish. Distention also helps to map out the whole stomach and to separate it from surrounding viscera.

PALPATION. Palpation of the stomach is closely associated with auscultation, inasmuch as the former elicits sounds (succussion, gurgling) which are helpful in diagnosis. (See Auscultation.)

But palpation elicits information independently of auscultation, chiefly in conditions of disease. Epigastric pulsation is common in anæmia; in nervous dyspepsia; in valvular disease of the heart, particularly tricuspid regurgitation, producing a liver pulse; and, more rarely, in aneurism of the abdominal aorta.

Increased resistance may be due to the hypertrophy of the muscular coat which coexists with distention of the stomach. When the stomach is shrunken and the resistance increased, it may be due to a diffuse carcinoma of the walls of the stomach; or rarely, to the so-called "fibroid stomach," the atrophy and thickening of the walls being due to chronic gastritis.

Increased resistance limited to the pylorus is found in carcinoma. The same effect produced by a tense right rectus muscle must be eliminated.

Position of Tumor. Cancers of the pylorus are situated usually between the xiphoid cartilage and the umbilicus, frequently a little to the right of the median line. But they may be found below the umbilicus, and exceptionally still lower down. Adhesions to neighboring organs commonly prevent the tumor from being moved.

When it has formed adhesions to the liver or diaphragm it moves with respiration.

From the statistics of Welch, based upon 1300 cases of gastric cancer, it appears that a tumor occupies the pyloric region in 60.8 per cent., or in three-fifths of all cases, the cardiac orifice being the next most frequent seat (11.4 per cent.); while in 80 per cent. of all cases a tumor is present. As a rule, tumors due to gastric cancer are small, hard, and irregular, and gradually increase in size.

Other non-malignant tumors are occasionally found, and also tumors due to adhesions around old ulcers and to puckered scars. The latter are distinguished from cancerous tumors not by the sense of touch but by their duration and clinical history.

The most exact method of determining the position and size of the stomach is by internal exploration combined with external palpation. A bougie is introduced into the stomach and swept over its entire internal surface, the position of the bougie being followed from point to point by the palpating hand.

This method is not advisable when it is possible to make a diagnosis without it, and is contra-indicated, according to Boas, by the following general diseases: Heart disease with failing compensation; angina pectoris; aneurisms of large vessels; recent hemorrhages of whatever kind; phthisis in progressive stage; emphysema with bronchial catarrh in progressive stage; apoplexies, complete or incomplete; hyperæmias of the brain; pregnancy; continued or remittent fever; great cachexia.

It is also contra-indicated by the following diseases of the stomach:

Ulcer with antecedent hæmatemesis or black stools; dilatation of stomach with typical vomiting; palpable cancer of pylorus, with emaciation, coffee-ground vomit and the other classical symptoms of cancer; in many neuroses of the stomach in which the character of the disease from the rest of the symptoms is clear; in acute gastric or intestinal catarrh associated with fever; when the mucous membrane of the stomach bleeds easily. Slight capillary hemorrhages constitute no contra-indication.

It will be seen from the above list that the method has a limited range of applicability.

Pain and Tenderness. Tenderness is elicited by palpation in gastritis, in dyspepsia, especially the catarrhal form, in ulcer, and in cancer. In gastritis and dyspepsia the tenderness is usually diffuse and is not constant; in cancer the tenderness is usually limited to the seat of tumor, but is not so marked nor so sharply localized as in ulcer. In ulcer tenderness is rarely absent, even when there is no pain, is very decided, and is so localized sometimes that it can be covered with the tip of a finger. Pain in the stomach from ulcer is chronic, circumscribed, and variously described as burning and wound-like. It is aggravated by palpation and food or drink, especially hot stimulating drinks, and relieved by cold, soothing drinks. It is accompanied frequently by pain in the corresponding vertebræ.

Diffuse pain is met with in acute and chronic gastritis, and in cancer of the stomach walls.

Percussion. *Position of the Stomach.* The stomach does not occupy a fixed position, and is a distensible organ. It is depressed by downward pressure of the diaphragm in deep inspiration, by emphysema, left pleural effusions, enlargements of the liver and spleen, and tight lacing; and raised by any causes which greatly distend the bowels or peritoneal cavity—tympanites, peritoneal effusions, tumors, etc. Moreover, after food is taken the stomach is distended and its position changed, being rotated anteriorly from below, the greater curvature rising and looking more forward, while the anterior surface has a more upward presentation.

The cardiac orifice of the stomach is fixed by its passage through the diaphragm and by peritoneal attachments which it receives there. It is behind the sternal insertion of the left seventh rib. The pylorus, on the contrary, is freely movable when the stomach is empty; it is nearly in the median line, but when the stomach is full it is pushed several inches to the right; it lies between the right sternal and parasternal lines on a level with the tip of the xiphoid cartilage.

Obrastzow (*Deut. Arch. für klin. Medicin*, Bd. xliii., 5, 417–456) divides the space between the navel and the xiphoid cartilage into three equal parts, and says that the *lower border* of the stomach, both in men and in women, is in the lower supra-umbilical third.

In children under fifteen the lower border rarely extends to the umbilical line; after fifty, on the contrary, it often extends below the navel. In conditions of bad nutrition it falls nearly to the navel.

According to Pacanowski and Wagner the *upper border* of the stomach, in the left parasternal line, lies at the lower border of the fifth

rib or in the fifth intercostal space, rarely at the fourth rib or in the sixth intercostal space. In the left nipple line it lies from the fifth interspace to the sixth rib, occasionally in the fourth interspace or at the seventh rib. In the anterior axillary line it lies at the lower border of the seventh or eighth rib, rarely above the sixth rib, never under the eighth rib.

A part of the anterior portion of the stomach and its lower border can be determined by percussion. Ordinarily, the most suitable position for examining the stomach is the recumbent one, with the knees drawn up so as to relax the abdominal muscles.

The stomach contains air at all times, but the amount varies greatly. The percussion note is tympanitic, frequently with a metallic ring; its quality is peculiar—"stomach tympany."

The percussion area of the stomach is *increased*, first, by causes external to the stomach; contraction of the liver, old pleurisy with retraction of lung, emphysema, former pregnancies, bad nutrition, and tumors pulling down the stomach; second, by intrinsic causes; distention or dilatation of the stomach.

Conversely, the percussion area is *diminished* by causes external to the stomach; enlargement of the liver and spleen, left-sided pleural effusion, pneumothorax, and hypertrophy of the heart.

Actual *diminution in size* of the stomach itself is difficult to demonstrate clinically with certainty. If upon inflation the great curvature remains at a higher level than 3 to 5 cm. above the umbilicus, diminution in size is highly probable. But even then the lower border may be prevented from descending by adhesions to surrounding viscera.

Enlargement of the stomach is generally due to *dilatation*, and is best marked clinically by a low position of the greater curvature. Dilatation of the stomach, according to Boas, can be separated from descent of the organ only when the greater curvature is more or less below the level of the navel, and when the greatest height of the stomach exceeds 10–14 cm. (4 to 5½ inches). But descent and dilatation are frequently present together.

In order to separate stomach tympany from that of the colon, which resembles it, the stomach may be distended with gas, while the colon contains solid or liquid matter; or if the colon be filled with gas the patient may be allowed to stand and to drink a glass or two of water. In either case the contrast between a dull and a clear note marks the boundary between stomach and colon.

Ziemssen recommends carbonic acid (developed by mixing sodium bicarbonate and tartaric acid) to distend the stomach; the quantity employed for adult men is seven grammes of bicarbonate of soda and six grammes (one and one-half drachms) of tartaric acid. Adult women should receive one gramme less of each.

As carbonic acid sometimes causes an uncomfortable oppression ordinary air is preferred by some. It can be forced in by a hand-bulb syringe attached to an ordinary stomach-tube. The percussion note over tumors of the pylorus is imperfectly tympanitic. Welch describes it as "tympanitic dulness." Less frequently it is dull, and rarely it is flat.

Traube has called special attention to the left lower portion of the thorax which projects over the stomach, "the halfmoon-shaped space." In health it gives a tympanitic note, unless the stomach or transverse colon is full or the omentum very fatty. In left pleural effusion it is dull. (See Diseases of Lungs.)

Auscultation. Auscultation can determine whether or not there is obstruction at the cardia. On listening over the œsophagus with the stethoscope when the patient is swallowing a liquid, a spurting sound is heard, followed in from five to ten or twelve seconds by a second sound which marks the escape of the fluid from the cardiac orifice of the œsophagus into the stomach. When there is obstruction of the cardiac orifice the second sound may be delayed as long as a minute.

When the stomach is partly filled with fluid a succussion or splashing sound can be produced by moving the patient quickly from side to side, or by quickly compressing the stomach and allowing it to rebound again immediately. Such sounds are abnormal if they are heard long after digestion should be completed and the stomach empty. The ear need not be applied to the body, but kept near by while the movements are made.

Normally, after drinking fluids a splashing sound is not developed lower than the umbilical line. If it is heard below this it is an indication of dilatation or of deep position of the whole stomach. Dilatation is very probable if the splashing sound is heard below the navel in a fasting stomach.

Furthermore, this sound is a sign of atony. If 50 to 100 grammes of water be swallowed, no splashing sound is heard unless there is atony of the stomach walls; but, if the atony is pronounced, a smaller quantity will be sufficient to develop the sound.

Examination of the Stomach by Chemical Methods. These methods have for their object the determination of the absorptive, motor and digestive energy of the stomach; the character of its secretions and their quantity; and indirectly to supply information bearing upon the presence or absence of atrophy, dilatation, and tumors.

Mode of Procedure. 1. Administer a test breakfast, as advised by Ewald (see page 491). 2. Remove the contents of the stomach one hour after breakfast is taken, by aspiration or by expression. *Aspiration* consists in the withdrawal of the stomach contents by suction, either with the ordinary stomach pump; by means of a bottle exhausted of air, as employed for paracentesis, and connected with the stomach sound; or by connecting the sound with a hand-ball aspirator.

Expression consists in introducing a sound and assisting the outflow of the fluid by pressing upon the epigastrium. If the tube is long enough it can be bent so as to assist expression with siphonage.

Aspiration is less disagreeable to the patient, and is necessary when the stomach contents are not fluid enough to flow easily, but it is subject to much the same contra-indications as obtain in the case of exploration of the œsophagus and stomach (see page 484).

Expression is not to be employed when there are old ulcers, ulcerating carcinoma, phthisis with antecedent hæmoptysis, or a disposition to menorrhagia.

These methods supply the most reliable information of the condition of the stomach and its secretions; because, when once withdrawn, their character can be ascertained accurately and the quantity measured; and, moreover, being able to choose the time of examination, we can decide whether or not what is found corresponds with health, and if not, in what particular it indicates disease. They permit a diagnosis to be made before other methods supply sufficient data.

A soft rubber tube, with two good-sized openings near its distal extremity, should be selected. Stockton suggests a tracing of rings around the tube one inch apart, beginning twenty inches from and ending thirty inches from the lower extremity. By means of the rings the length of tube inserted can be told. In healthy adults the distance from the incisor teeth to the lower border of the stomach is about twenty-two inches. In dilatation it may extend from twenty-four to thirty. The distance is partly determined by success in the siphonage. If the return flow of fluid does not take place, it is well to either withdraw the tube or push it further on, for if too long it may curve above the level of the fluid, or if too short it may not reach the fluid.

After the tube is oiled, or coated with the white of egg, the patient should be seated, and the tube at once passed to the back of the pharynx, and, with or without guiding by the finger, pushed toward the œsophagus. It is at once grasped by the œsophagus or lower pharynx, and if the patient is instructed to swallow and to breathe slowly it is rapidly carried downward by deglutition. Mucus that accumulates in the mouth after the tube is passed should be allowed to dribble outward, and not be swallowed. It is often of advantage to assure the patient by having him pronounce the letter "a" or some small syllable. It is not necessary to extend the head backward.

If a hard tube is used, it must be guided by the operator, who should stand back of the patient supporting the head, which has been thrown backward. The tube can be passed if the operator is seated in front of the patient. This kind of tube is used with the stomach pump.

Characters of Normal Gastric Contents. The amount of fluid, after digestion of the test breakfast has continued for one hour, is from 30 to 40 c.c. After filtering, the filtrate is clear, yellow or yellowish-brown in color. If the digestion is normal, the fluid should contain free hydrochloric acid, and no lactic acid. It should also contain pepsin, rennet (the milk-curdling ferment) and organic acids. Albuminoids should be converted into peptones, and starches into achroödextrin, dextrose, or maltose.

Chemical and Physical Examination. The following steps are necessary, and a description of them will be given in due course. 1. The acidity is determined by litmus paper. 2. The odor. 3. Inspection is employed (*q. v.*). 4. The presence of free acid, of HCl, of lactic acid, of acetic acid, and of butyric acid are determined. The amount of total acidity is then estimated in order to judge of the amount of secretion of the gastric juice or hydrochloric acid. 5. Tests are then made to determine the presence of pepsin and pepsinogen, bearing in mind that if hydrochloric acid is present in a free state these constituents are sure to be present. 6. The test for the milk-

curdling ferment is also made. 7. Having determined the chemical nature of the filtrate, examination should be made to determine the degree of progress of the digestion of the proteid and the carbohydrate elements of the food by testing for proteids, as serum-albumin and peptone, and by testing for starch and its products. It is to be observed that perfect familiarity with normal digestion, and particularly the proper length of time required to perform definite acts, is very essential.

Reaction. The reaction of the contents of the stomach is usually acid, from the hydrochloric acid of the gastric juice. It may be alkaline in cases of hemorrhages, or in the vomiting known as water-brash.

Odor. The odor is sour normally, but it may be aromatic from the presence of the fatty acids, fæcal in obstruction of the bowels with fæcal vomiting, and, finally, may indicate the nature of poisonous ingesta —ammonia, phosphorus, carbolic acid.

Inspection of the Stomach Contents. The contents of the stomach may be obtained by emesis or by aspiration. The latter is preferable. They should be inspected, first, as to *quantity.* If a person has taken no food or drink between the evening meal and the following morning the stomach should not contain more than three and one-half fluid ounces; more than this is abnormal. The *character* of the stomach contents is important. If undigested food is found after digestion normally should be completed, then there is deficient digestive energy. No undigested food should be found longer than six or seven hours after an ordinary meal of mixed foods, and the stomach should be empty much sooner if only starches are taken, as in Ewald's test breakfast.

Mucus is found in small quantity normally, but is increased in catarrhal affections of the mouth, throat, or stomach. When its source is the mouth, *saliva* also is generally present.

Bile and intestinal juice may be regurgitated into the stomach as the result of violent emesis, or when the pylorus is much relaxed, or in stenosis of the duodenum below the common duct; bile is then present in large quantity if the stomach is dilated.[1] Bile is recognized by the usual tests (see under Examination of Urine), and intestinal juice by its peculiar properties and the presence of leucin and tyrosin.

Blood is found in ulcer; cancer; acute, especially toxic, gastritis; injuries to the mucous membrane from the use of the sound for expression, and violent retching. It is also common in cirrhosis of the liver, and may occur in purpura, peliosis rheumatica, the hemorrhagic diathesis, and in yellow fever.

If the blood is unaltered it can be distinguished from all other substances by microscopic examination. Occasionally the blood has the appearance of coffee-grounds. The *hæmin test* serves to distinguish it. The suspected material is filtered and a little of the filtrate evaporated in a watch-glass; when dry a small portion is mixed with finely pulverized salt upon a glass slip; it is then covered with a cover-glass and one or two drops of glacial acetic acid allowed to flow under the cover-glass. The acetic acid is evaporated by slowly heating the slip over a

[1] Hochhaus: Berlin. klin. Woch., No. 17, 1891.

small flame, and when dry a few drops of water are allowed to flow under the cover-glass to dissolve the salt. If the vomit contained blood, brown rhombic crystals of hæmin (hydrochlorate of hæmin) will appear under the microscope. As they are very small, a magnification of about 300 diameters will be necessary to bring them into easy view.

Pus is rarely present in sufficient quantity to be detected by the naked eye, but it sometimes occurs in phlegmonous gastritis and when an abscess has ruptured into the stomach. In microscopic amounts it may be found in severe catarrhal affections.

Fœcal matter is vomited in complete obstruction of the bowels, and, according to Vierordt, in severe diffuse peritonitis. It is recognized partly by its appearance and partly by its odor.

Worms are sometimes vomited; the round worms not so very unfrequently; oxyurides and ankylostomata rarely.

Digestive Energy. Inspection of the vomited matters, or the contents of the stomach removed by aspiration, shows whether there has been digestion or not, and what variety of food, albuminoids or hydrocarbons, has been undigested. Boas states that an abnormally great quantity of solid matter and small amount of chyme indicates an abnormal retention of the latter, which is usually brought about by motor weakness (atony, dilatation of the stomach), or dilatation in conjunction with deficient absorptive power. Not rarely when there is a large residue in the stomach the contents separate into three layers. The uppermost is mucus or undigested food; the second, generally the thickest layer, consists of fluid; and the lowest layer is chyme. Such a formation, he says, points to abnormally long retention as the result of stenosis and consecutive dilatation, or to motor weakness.

One hour after the administration of a test breakfast of 35 grammes of white bread and 300 grammes of water there should remain 40 c.c. Hence if, after such a breakfast, there is found a much greater quantity, then motor or absorptive insufficiency may be considered to exist. A filtrate of 100 to 300 c.c. is very probably due to organic obstruction to the outflow, stenosis of the pylorus, adhesions, or dislocation of the pylorus. Of course, to be sure that the stomach contains nothing at the time of giving the breakfast, it must first be emptied.

When the stomach has retained its contents a long time, as in dilatation, so that fermentation has taken place, sarcinæ and torulæ may be found.

Tests for the Presence of Acids. Normally lactic acid is found during the first half-hour of digestion, when starches have been taken. When only meats have been taken lactic acid is not found. The secretion of hydrochloric acid is not delayed until then, but is at first combined, and cannot be detected as free acid until half or three-quarters of an hour afterward. The stomach contents, when aspirated or expressed, should be filtered before testing.

Free acids. The most sensitive test for free acids is Congo red. Filter paper soaked in it and allowed to dry is turned a light greenish-blue by HCl, and a darker blue by lactic acid. Wolff[1] was able to

[1] Trans. Phila. Co. Med. Soc., 1889, x. 305.

detect one part of HCl in 20,000 parts of water. When no reaction is obtained, therefore, entire absence of acidity may be assumed.

Free HCl. *Tropæolin* 00 is declared by Boas to be an absolutely certain test for HCl. A saturated alcoholic solution is of an orange-yellow color. Three or four drops of it are placed in a white porcelain dish and spread upon the sides of the dish by rotating it. The same amount of the fluid to be tested is then allowed to trickle down the sides of the dish and to be intimately mixed with the tropæolin. Upon heating the dish over a small flame, splendid lilac-blue to blue streaks, characteristic of HCl, will appear if that acid is present. No organic acid gives the same color.

Tropæolin paper is turned brown by gastrice juice containing HCl, the brown changing to blue upon the paper being heated. Organic acids give a brown color also, but it disappears upon heating.

Phloroglucin vanillin, introduced by Günsburg, is also a very sensitive test for HCl. The following combination is said by Boas to be more sensitive than the ordinary one, which contains only 30 grammes of absolute alcohol:

Phloroglucin	2.0 (gr. xxx)
Vanillin	1.0 (gr. xv)
Alcohol (80 per cent.)	100.0 (f ℥ iij)

Three drops are put into a porcelain dish and an equal quantity of the stomach filtrate. Upon *cautious* heating over a *small* flame a beautiful carmine surface is formed, especially at the edges. The same color is not produced by organic acids. Filter paper soaked in it and moistened with a few drops of stomach filtrate, containing HCl, changes on heating to a beautiful carmine, which is unaltered upon the addition of ether.

Test for Free HCl. Boas' method is a modification of that of Mintz. Ten c.c. of the gastric fluid are shaken with 100 c.c. of ether until organic acids are removed. The Congo-red test is then employed until the grayish-blue discoloration cannot be secured.

In testing for the presence of HCl, it is better to give the patient a meal which is known to be digestible within a certain time by stomachs in a normal state, otherwise HCl may appear to be absent because it is still combined with albuminoids.

Ewald's test breakfast is the simplest. He gives in the morning, on an empty stomach, one or two ounces of bread and a cup of tea or an equivalent amount of water. In one hour the contents of the stomach may be aspirated and tested for HCl.

Amount of Free HCl. If by previous tests HCl is found alone, its percentage is easily calculated. To a measured quantity of the gastric fluid add drop by drop from a burette deci-normal alkaline solution until the acid is neutralized. One c.c. of the alkaline solution is equivalent to 0.003646 HCl. Multiply the number of c.c. required to neutralize 10 c.c. of the gastric solution by 0.003646, and again by 10, the result will be the percentage of acidity. If 6 c.c. are used the percentage will be $6 \times 0.003646 \times 10 = 0.218$, within the normal range, or from 0.14 to 0.24 per cent. Günsburg's test can be used to estimate

the quantity of HCl. This is employed by diluting the stomach contents until the test is not responded to. In health the limit of response is found when one part of HCl is found in 20,000 parts of the fluid. In abnormal conditions, when the gastric fluid is diluted, one-half the proportion is 2 to 20,000, or 1 in 10,000. If the fluid is diluted to 10 times its original strength, it is 10 to 20,000, or 1 in 2000.

Significance of HCl. If absent one to three hours after taking milk or nitrogenous food, there is serious impairment of function. An increase to 0.33 per cent. does not imply functional disorder. Absence occurs in cancer and chronic gastritis; an increase in ulcer or in neuroses.

Presence of Lactic Acid. If this continues an hour after the test meal it is pathological. Its presence may be determined by Uffelmann's reagent: Mix one drop of pure carbolic acid with five drops of a dilute solution of neutral ferric chloride. Add sufficient water to render the whole of an amethyst-blue color. In this add a few drops of the gastric fluid. A mere trace of lactic acid will change the blue to a light yellow.

The test for lactic acid is simulated when phosphates, glucose or alcohol is present in the gastric juice. The lactic acid should be removed by extracting with ether.

The Fatty Acids. Butyric acid is detected by the same reagent. It strikes a tawny yellow color with a reddish tinge. As much as one part of the reagent in 2000 is required.

A few c.c. of the filtered gastric fluid is shaken with three or four times the amount of ether. The ether is poured off when it rises on the top, and fresh ether added and the washing repeated. After the third washing the ether that cannot be poured off is evaporated through a water bath. Add a few drops of water to the residue and then an equal quantity of the reagent. The characteristic color is produced.

The Total Acidity. This is determined by titration. Fill a Mohr's burette with a deci-normal solution of caustic soda. To 10 c.c. of the filtered gastric fluid add two drops of an alcoholic solution of phenol-phthalein. Allow the caustic soda solution to drop slowly from the burette into the fluid, until the red color which is produced does not disappear on shaking. The color is produced by the action of the alkali on the phenol-phthalein. Four to six c.c. of the caustic soda solution are required to neutralize the acid in normal digestion. The degree of acidity is expressed in percentage. Thus if four c.c. neutralize ten c.c. the total acidity will amount to 40 per cent., or if six c.c. are required, to 60 per cent. of the normal.

If more or less than the amount just indicated of the alkaline solution is required to neutralize the acid, the total acidity is increased or diminished, and hence is abnormal.

Test for Fatty Acids. In addition to Uffelmann's test the fatty acids may be detected by boiling a few c.c. in a test-tube over the mouth of which blue litmus paper is attached. If acid is present its vapor will change the blue to red. Particles of pure fat may be seen floating in the gastric contents, or they may be extracted by ether. *Acetic acid* is recognized by its odor, particularly after heating the solution. It

may be detected as follows: Secure an ethereal extract of the gastric contents, evaporate in a water bath, and dissolve the residue in water. Neutralize the watery solution with sodium carbonate, and then add neutral ferric chloride solution. A blood-red color results if acetic acid is present.

Alcohol is detected by its odor, and by Lieben's iodoform test. Distil the stomach contents, add to a portion a small quantity of liquor potassa, and then a few drops of iodine-iodide of potassium solution. A precipitate of iodoform takes place slowly if alcohol is present. If acetone is present it forms rapidly.

Test for Pepsin. If HCl is present add 5 c.c. of a gastric filtrate to a small piece of egg-albumin. Allow digestion to take place for several hours at 37° to 40° C. Non-digestion indicates absence of pepsin.

If HCl is absent pepsinogen is found alone. Add two drops of a 25 per cent. HCl solution to 10 c.c. of the gastric contents. Add to this solution a small portion of egg-albumin. If it is dissolved, pepsinogen was converted into pepsin by HCl.

Test for Rennet (the milk-curdling ferment). This may be detected as follows: From 5 to 10 c.c. of cow's milk of neutral reaction is boiled and added to neutralized and filtered gastric juice. Place the mixture on a warm bath heated to 30° to 40° C. The casein of the milk is precipitated in flakes in from 20 to 30 minutes if the ferment is present.

Test for Carbohydrates. Add a few drops of Lugol's solution to the gastric contents. If starch is present it turns blue. If erythrodextrin, it becomes purple. If the digestion has proceeded so far as to change starch into dextrose, the iodine hue remains unchanged. The starches should be completely digested an hour after they are taken into the stomach, hence in health the iodine hue should not change after this time.

Test for Peptones. If the albumin has been converted into peptone a distinct purplish-red color is struck when a small amount of caustic potash and a little dilute cupric sulphate are added together. If there is albumin or syntonin the color is violet blue.

Günsburg's Test of Digestive Energy. Günsburg has introduced the use of iodide of potassium in the following way: From three to five grains are placed in a rubber tube of extremely thin walls; the ends of the tube are then bent and brought into apposition and fastened in that position with three fibrin threads made firm by preservation in alcohol. The whole packet is then pressed into an empty gelatin capsule and given to a patient to swallow one-half hour after a test breakfast. The saliva is tested for iodine every fifteen minutes. The more rapid the solution of the capsule and fibrin threads, the sooner the iodine can be absorbed and appear in the saliva, and hence this rapidity is an index of the digestive energy.

The method is liable to fallacies: solution of the fibrin may take place in the intestine instead of the bowel, and the threads may be loosened by the acids of fermentation instead of by digestion. Nevertheless the test is a valuable one, especially when aspiration is inadmissible.

Test of the Absorptive Energy of the Stomach. Penzoldt and Faber recommend the administration of three grains of chemi-

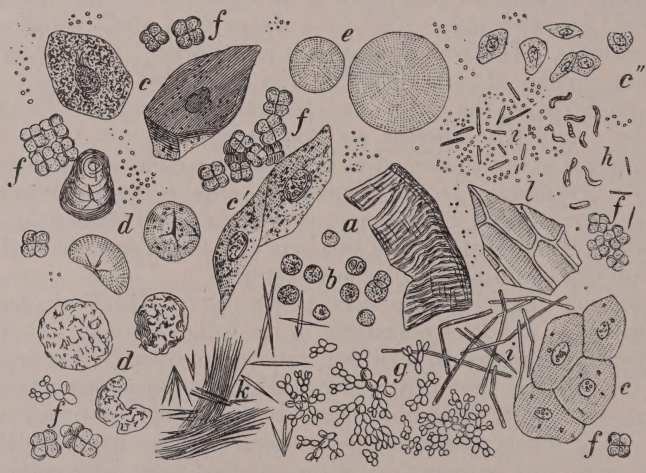
cally pure iodide of potash, *i. e.*, free from iodic acid, a short time before dinner. Any fragments of free iodine adhering to the iodide of potash are first carefully washed away. The saliva is tested for iodine with starch-paper and fuming nitric acid. If absorption is active a violet color is obtained in from six and one-half to eleven minutes, and a blue color in from seven and one-half to fifteen minutes. The character of the food taken is said to have considerable influence in retarding the appearance of the reaction, so that the blue reaction may not appear for forty-five minutes.

Boas states that in dilatation of the stomach the reaction may be delayed to two hours, and in cancer as long as eighty-two minutes.

Test of the Motor Power. Ewald and Sievers have suggested the use of salol; fifteen grains are given, and normally salicylic acid should be detected in the urine in from forty to sixty minutes, or in seventy-five minutes at the latest. If it is deferred still longer, motor insufficiency is indicated. Urine containing salicylic acid gives a dark, brownish-red color upon the addition of a drop of tincture of the chloride of iron.

Microscopical Appearance of Vomit. The illustration from Von Jaksch shows the various matters which may be found in vomited

Fig. 79.



Collective view of vomited matter. (Eye-piece III., objective 8 A, Reichert.)

a, Muscle fibres. *b*, White blood-corpuscles. *c*, *c'*, Squamous epithelium. *c''*, Columnar epithelium. *d*, Starch grains, mostly already changed by the action of the digestive juices. *e*, Fat globules. *f*, Sarcinæ ventriculi. *g*, Yeast fungi. *h*, Forms resembling the comma bacillus, found by the author once in the vomit of intestinal obstruction. *i*, Various micro-organisms, such as bacilli and micrococci. *k*, Fat-needles; between them connective tissue derived from the food. *l*, Vegetable cells. (Von Jaksch.)

matter. Briefly, they are columnar and squamous epithelium; white blood-corpuscles acted on by gastric juice; red blood-corpuscles. The corpuscles are usually isolated. The red are rarely perfect, and in the white little more than the nuclei remains. From the food we may also find muscle fibres, fatty globules and fat-needles, elastic fibres and

connective tissue, starch granules, and vegetable cells. Muscle fibres are recognized by their transverse striation. Fat globules are soluble in ether, and are recognized by their refracting powers. Starch granules stain blue, with iodo-potassic-iodide solution.

In addition, fungi of many forms are found, as the mould fungi; the yeasts, and fission fungi. The latter are recognized after staining by the iodo-potassic-iodide solution, which colors them blue. The most important fission fungi are the sarcinæ ventriculi. They are of a dark gray tint, stain mahogany-brown to reddish brown with the above-mentioned solution, and resemble in shape bales of wool. (See Bacteriological Diagnosis.)

Gastric Hemorrhage. Hemorrhage of the stomach, *hæmatemesis*, or vomiting of blood, is due to an organic lesion, or the effects of acute irritant poisoning. The blood is vomited. Care must be taken to see that the vomited blood is not from the upper air-passages, and previously swallowed. If the hemorrhage is profuse, the blood may cause irritation of the larynx, and provoke paroxysms of coughing. It is often difficult, therefore, to distinguish hemorrhage from the lungs and hemorrhage from the stomach.

Hæmatemesis.	Hæmoptysis.
1. Previous history points to gastric, hepatic, or splenic disease.	1. Cough or signs of some pulmonary or cardiac disease precedes, in many cases, the hemorrhage.
2. The blood is brought up by vomiting, prior to which the patient may experience a feeling of giddiness or faintness.	2. The blood is coughed up, and is usually preceded by a sensation of tickling in the throat. If vomiting occurs, it follows the coughing.
3. The blood is usually clotted, mixed with particles of food, and has an acid reaction. It may be dark, grumous, and fluid.	3. The blood is frothy, bright red in color, alkaline in reaction. If clotted, is rarely in such large coagula, and muco-pus may be mixed with it.
4. Subsequent to the attack the patient passes tarry stools, and signs of disease of the abdominal viscera may be detected.	4. The cough persists, physical signs of local disease in the chest may usually be detected, and the sputa may be blood-stained for many days. (Osler.)

The hemorrhage may continue within the stomach without exciting vomiting. The general symptoms of hemorrhage may appear first, as pallor, dimness of vision, giddiness or fainting. The blood which comes from the stomach is usually acted upon by the gastric juice, and is dark, clotted, and partially digested. It is often mixed with food. It is acid in reaction. In large hemorrhages the blood may be fluid, and of a scarlet color, but if retained for any length of time is coagulated. The vomited matter has the appearance of coffee-grounds, when there is a small amount of blood. When large in amount, and digested, it appears like tar.

Vomiting is usually followed by movements of the bowels. The matter discharged is of characteristic appearance. It is black or tarry. It is distinguished from hemorrhage of the intestinal canal below the duodenum by the color of the blood. In intestinal hemorrhage from this situation, the blood is distinctly red. The dark stools must not be confounded with the same character of stools seen when iron or bismuth is taken. In rare instances a hemorrhage of the stomach may take place because of disease of the lower part of the œsophagus.

Causes. 1. General diseases from changes in the blood cause gastric hemorrhage, as scurvy, purpura, hemorrhagic smallpox, yellow fever,

acute yellow atrophy of the liver, and in severe anæmia, leukæmia, Hodgkin's disease, and pernicious anæmia. 2. Ulcer of the stomach. 3. Cancer of the stomach. 4. Ulcer of the duodenum. 5. Portal congestion, as in cirrhosis of the liver, and other forms of chronic hepatic disease; disease of the spleen. 6. Congestion due to disease of the heart. 7. In chonic Bright's disease with atheroma. 8. Rupture in aneurism. 9. Vicarious menstruation.

Profuse and sudden hemorrhage, in the absence of well-marked symptoms of disease, is in nearly all cases due, either to latent ulcer, or to congestion of the stomach from early cirrhosis of the liver.

Data Obtained by Inquiry. The Subjective Symptoms of Diseases of the Stomach.

The patient suffering from gastric disorder will be likely to complain of one or more of the following symptoms: Disorder of the appetite, bad taste in the mouth, thirst, eructations, pyrosis, distress, weight and burning after meals, flatulency, nausea, vomiting, constipation, diarrhœa, pain, vertigo, and cardiac palpitation. The subjective symptoms are detailed in the section on gastric neuroses.

Bad taste in the mouth, with a heavy breath, is usually due to acute catarrh; it may be present in chronic catarrh. It is said to be characteristic of the acute form of gastritis popularly known as biliousness.

Thirst is not a symptom of gastric disorder alone; it is a symptom of diabetes and all conditions in which the body has lost fluids, as water by vomiting or purging, blood by hemorrhage, or water by evaporation and combustion (fever). It is common in acute and chronic gastritis, particularly in the alcoholic form.

Distress, weight, and burning. They are frequent complaints. They exist in varying degrees, and may be single or combined. (See Gastric Hyperæsthesia.)

Nausea. This symptom is usually associated with vomiting. In some persons it is impossible to excite vomiting, although they may suffer intolerably from nausea. Nausea is akin to vomiting in its mechanism and clinical associations (*q. v.*). It is a common incident in chronic interstitial nephritis. In old people, with arterial sclerosis and defective renal elimination, it is common. It may be due to irritating ingesta, to hyperacidity, to gastrectasia, or to toxins formed within the stomach.

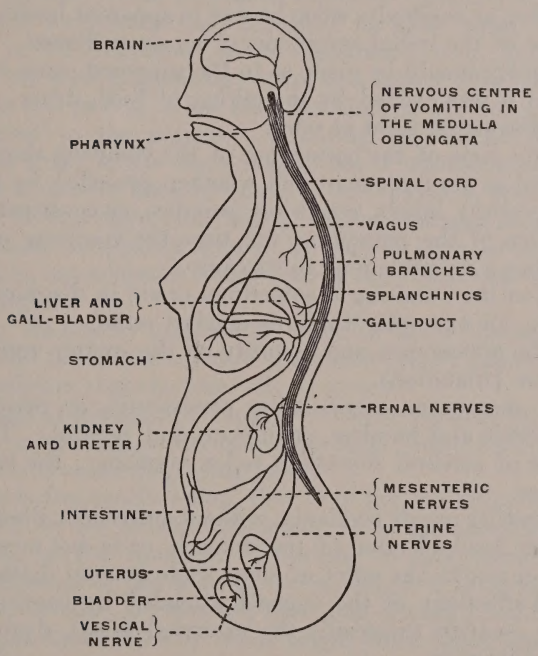
Vomiting. Vomiting takes place when the stomach is compressed by the abdominal muscles and diaphragm, coincidently with relaxation of the so-called cardiac sphincter of the œsophagus. Sometimes there are nausea and violent efforts at expulsion on the part of the stomach, but no vomiting occurs because the cardiac orifice of the stomach is not at the same time opened. Again, there may be profound relaxation of the œsophagus, but no compression of the stomach by the diaphragm and abdominal muscles. Both factors must operate at the same time to result in vomiting. This explains why it is that some persons suffer extreme nausea and have even violent retching, but are unable to vomit.

It is to modern physiologists—Schiff and Budge and Brunton—that we owe a correct explanation of the physiology of vomiting.

From them we learn that there is a nervous centre for vomiting, which is seated in the medulla oblongata, in close proximity to and intimately connected with the respiratory centre. It is to this centre that impressions are sent from the brain itself or from various portions of the body by their nerve supply, and from this centre that motor impulses are transmitted to the muscles concerned in the act of vomiting, and to the stomach and œsophagus. In his usual graphic manner, Brunton has described the entire mechanism.

By a very good diagram (see Fig. 80) the author indicates the afferent nerves which transmit impulses to the vomiting centre, exciting it to

FIG. 80.



action. They are: pharyngeal branches of the glosso-pharyngeal; pulmonary branches of the vagus; gastric branches of the vagus; gastric branches of the splanchnic; renal, mesenteric, uterine, ovarian, and vesical nerves. Fibres pass downward from the brain, conducting impressions to the vomiting centre from the organs of special sense, from the brain substance or its membranes when the seat of disease, or from central ganglia excited by emotion or imagination.

From this it is seen that vomiting is a reflex act; that its mechanism is quite simple; and that a proper understanding of this mechanism is essential to a correct appreciation of its pathology and treatment. Reference has not been made to the vomiting that occurs in the initial stage of

many fevers, and in septicæmia, uræmia and allied affections, and to the vomiting of hysteria. In the former it is doubtless due to the direct action of the poisoned blood on the centre, but it can also readily be seen to be due to the propagation of impulses to the centre from the brain that is irritated by the blood. If the phenomena of hysteria are due to an abeyance of the processes of inhibition, the occurrence of vomiting can be said to arise from the non-control, by higher centres, of this centre. (From "Vomiting, Physiological and Clinical," *Trans. Penna. State Med. Soc.* Musser.)

The significance of vomiting in a given case can sometimes be determined very readily, and sometimes it remains in doubt after very careful examination and questioning of the patient. In seeking for the explanation of vomiting it is of importance to find out the previous health of the patient; whether it occurred after the patient had been ill for a longer or shorter time, or suddenly, when he was in apparent health, or whether it formed one of the initial symptoms of an acute disease.

Again, inquiry should be made as to the supposed cause of the vomiting; whether it was excited by the taking of food, drink, or medicine, or by some disgusting sight or odor.

Further, the time of the occurrence of the vomiting should be ascertained, as well as its frequency, and whether preceded by nausea, pain (noting its locality), injury, coughing, jaundice, or constipation.

The position of the patient at the time the vomiting occurs sometimes furnishes a valuable clue to its cause.

The effect of the vomiting is sometimes of aid in diagnosis. In ulcer and migraine, for example, it affords marked relief.

Finally, the appearance and quantity of the matter vomited is very important (see Dilatation).

Vomiting may occur occasionally, persistently, or periodically. It may be projectile and painless, or difficult and painful. The former is characteristic of cerebral disease or reflex vomiting; the latter of local gastric disease.

When vomiting occurs suddenly, without antecedent illness, it usually indicates some local affection of the stomach, or is due to some nervous impression, or marks the onset of some acute general disease.

The local affections of the stomach attended by vomiting are acute and chronic gastritis (especially the catarrhal form), dyspepsia, ulcer, cancer, and dilatation.

In *acute gastritis* there will be a history of an acute illness marked by decided local and general symptoms. The cause of the gastritis may be found to be over-eating of highly seasoned or indigestible food; abuse of alcohol, narcotics, or sedatives; drinking water to which the patient is unaccustomed; poisoning with such drugs as arsenic and mercury; sudden changes in atmospheric conditions in susceptible persons.

The vomiting is preceded by nausea, gastric pain, and tenderness, and often followed by profound prostration.

The vomited matters consist, first, of the contents of the stomach (which may throw light on the cause of the attack), then of mucus, saliva (which has been swallowed), bile, and, in grave cases, altered blood.

In *chronic gastritis* vomiting often occurs in from half an hour to an hour and a half after eating, the food being only partly digested and sometimes coated with mucus. It does not produce the prostration that vomiting in acute gastritis does, and is followed by some relief to the gastric uneasiness and pain. The emaciation may suggest cancer of the stomach.

In *ulcer of the stomach* vomiting is rarely absent. It occurs usually soon after taking food, and its occurrence affords relief to the gastric pain. There is nothing characteristic in the vomit unless it contains blood. Welch thinks that gastric hemorrhage in recognizable amount occurs in about one-third of the cases.

In *cancer of the stomach* vomiting is an almost constant symptom, but it may not occur until comparatively late in the disease, or, more rarely, may be one of the earliest symptoms. Usually it appears first after dyspeptic symptoms have persisted for some time. There is no uniformity in the frequency of its occurrence or in the character of the vomit. As a rule, vomiting occurs at a longer interval after taking food than in the case of ulcer, and the ejection of food does not give as much relief to the patient. Vomiting may occur every day or several times a day in the early stages, but if *dilatation* of the stomach develops, as it usually does in cancer of the pylorus, vomiting may be deferred for several days, and then be correspondingly more copious in amount. Blood, frequently altered by gastric juice so as to resemble coffee-grounds, is a common constituent of the vomit (see under Inspection—Vomit).

Vomiting frequently marks the onset of acute diseases, especially *pneumonia* and the *eruptive fevers* and *yellow fever*. Marked vomiting generally indicates that the case will be severe.

Nausea and vomiting are excited in some persons by the *sight* of blood, or by a horrible or loathsome spectacle; others are more susceptible to foul *odors* and disgusting *tastes*.

Vomiting is frequently reflex, that is to say, irritation at some point is transmitted by the proper afferent nerve to the vomiting centre and then reflected to the stomach. Vomiting of this character occurs in *pregnancy*, diseases of the *ovaries*, *uterus*, *bladder*, *prostate*, *lungs*, *nose*, *eyes*, *kidneys*, *intestine*, *peritoneum*, *liver*, *gall-bladder*, *and bile-ducts*.

Vomiting is found to be of reflex origin when there is no local affection of the stomach present, and no general disease to account for it, and when a remote source of irritation can be discovered, the removal or mitigation of which checks the vomiting. The particular organ which is the source of the irritation must be determined by a careful physical examination guided by the indications furnished by the age, sex, time of occurrence, habits and other symptoms which accompany the vomiting.

The nausea and vomiting from which many women suffer during the early months of pregnancy are most marked on rising in the morning; they are aggravated if the patient has been on her feet much or has been subjected to any exhausting or worrying influence; on the other hand, they are relieved by quiet and the recumbent posture. In diseases of the ovary, uterus, bladder, and prostate there are local pain, catarrhal symptoms, inflammation or enlargement to attract attention.

The lungs probably are not often the cause of reflex vomiting. Rarely, however, phthisis is so masked by gastric symptoms and vomiting that it may be overlooked. More frequently it is the act of coughing and the effort to expel the sputa from the throat which produces the vomiting. Expectoration tickles the throat and may play the part of the finger or feather in inducing vomiting. This seems to be the explanation of the vomiting which ensues upon a hard spell of coughing in pertussis.

Peritonitis may be suspected to be the cause of vomiting if there has been injury to the peritoneum from a surgical operation, or if it has been exposed to infection through the uterus and tubes, or from disease of organs surrounded by it, as the vermiform appendix. Vomiting may be the only symptom present excepting collapse. The fluid is not ejected alone, but regurgitated, and may seemingly pour out of the stomach. Large amounts of fluid are discharged, clear or of a green color.

In the vomiting due to the passage of a renal calculus or gall-stone the colicky pains and their location definitely point to the source.

Vomiting is also a marked symptom of diseases in which poisons circulate in the blood; they produce vomiting probably by direct irritation of the vomiting centre. Among such diseases are the *specific fevers*, notably *scarlet fever* and *yellow fever; sewer-gas poisoning;* diseases of the liver and kidney, which produce *cholæmia* and *uræmia*, particularly cirrhosis of the liver and interstitial nephritis.

The vomiting of *uræmia* usually occurs in the morning. It is accompanied by nausea and depression. Whenever morning nausea and vomiting occur in an adult without obvious local cause the urine should be examined. Other confirmatory signs are high-tension pulse, accentuation of the aortic second sound, and hypertrophy of the heart.

Vomiting due to *cerebral disease* is well recognized. In early life it is a characteristic feature of meningitis and tumor of the brain. It is likewise of moment in later life. I am of the conviction, however, that it is not sufficiently recognized as one of the first symptoms of apoplexy. True, we find apoplexy occur after a full meal, when the attack is associated with indigestion, with efforts at vomiting. It is not to these cases that reference is made, but to cases of painless, often watery vomiting, occurring without nausea and without retching. A sudden, violent propulsion of the stomach contents, ceaseless, unrelieved by remedial measures, has been seen by the writer to precede other signs of apoplexy by from thirty minutes to twenty-four hours. In all cases of an apoplectic character the pulse is slow and full, while in nausea and vomiting from other causes, in the aged particularly, it is weak and feeble. Moreover, some alteration of breathing is noticed. It is either irregular, or slow, or unduly hurried. It proves the intimate relation of the vomiting and the respiratory centres. Further, strength is seen, not weakness; in the apoplectics the face is congested, not pallid, as in simple sick stomach. The other peculiarities of cerebral vomiting have been indicated.

Diagnosis. Vomiting is readily recognized. It is often productive of serious symptoms. It may cause apoplexy or cerebral congestion; it may cause acute overdistention of a dilated heart, as in aortic regur-

gitation. If it continues any length of time and much fluid is ejected it is attended by anuria, and rapidly followed by collapse. Thirst is also produced.

Flatulency. Flatulency is caused by an accumulation of gas in the stomach or intestines. It is a very common source of complaint on the part of patients. Gastric flatulency is marked by a distention of the stomach, with the discomfort which it occasions, and by the eructation of gas at variable intervals after the taking of food. When the gas is the result of fermentation with the production of the fatty acids, flatulency is frequently accompanied by pain, which is relieved by eructations. When the distention is great or long-continued, disturbances in the action of the heart, particularly palpitation and intermittency, are liable to occur. Occasionally the breathing is interfered with, and, from the apprehension which this symptom and palpitation excite, faintness and inaptitude for mental and physical work may arise.

Excessive flatulency is a common manifestation of hysteria. Such patients may complain of something rising into the throat from the stomach and smothering them (*globus hystericus*). There may also be tympanites, and even phantom tumor. It may be necessary fully to anæsthetize the patient to diagnosticate the latter from genuine tumor.

Vertigo. The stomach is but one of a number of sources of the production of vertigo. Some patients find by experience that certain articles of food, such as oysters or lobsters, have to be avoided because they produce vertigo, although digestion is good, and more indigestible articles can be taken without inducing any such result.

In other cases acute indigestion from over-eating, particularly if it result in the development of an acid condition of the stomach, is apt to be accompanied by vertigo when the stomach symptoms are most severe. Usually the vertigo is associated with headache, more or less intense; it is relieved by closing the eyes and lying down, but does not wholly disappear until all the symptoms gradually subside after free vomiting. Some persons are subject to so-called "blind" headaches—headaches accompanied by dimness of vision, more or less mental confusion and uncertainty of gait, possibly with staggering and often with vertigo. Such headaches appear to be due to an acid condition of the stomach, and are relieved by alkalies or vomiting.

It is difficult to separate the vertigo of chronic gastric or gastro-intestinal dyspepsia from that of lithæmia or latent gout. Probably both are due not to any local irritation transmitted to the brain, but to the circulation in the blood of toxic products of digestion, which act upon the brain. The vertigo is not as severe as in acute indigestion or acute dyspepsia, but it is constant. With some patients it is associated with unconquerable timidity of walking alone upon the street.

Pain. *Cardialgia* is a form of discomfort in the epigastrium scarcely amounting to pain, but attended by heartburn or acidity. *Gastrodynia* is a violent pain spoken of as cramp or spasm of the stomach. The pain is transient. *Gastralgia* is a form of pain with features like that of neuralgia, occurring when the stomach is empty. (See Gastric Neuroses.)

Pain referred to the stomach is situated in the upper zone of the abdomen, below the ensiform cartilage, between the ribs of the two sides,

usually in the median line. It may be along and under the left ribs. Pain in this situation may be due to a number of causes. 1. Pain from myalgia, neuritis, or neuralgia of the intercostal nerves, which terminate in this situation. (See Abdominal Pain.) 2. Localized peritonitis or perigastritis, a cause for which may be found in the occurrence of infection or injury of the peritoneum from disease of contiguous organs. 3. Affections of the pancreas may cause pain. *a.* Pancreatic colic, a rare condition associated with diarrhœa, intestinal dyspepsia, and salivation. The pain is paroxysmal, the attacks lasting two or three hours. *b.* Pain due to carcinoma of the pancreas, darting or lancinating in character, associated usually with tumor, jaundice, and emaciation. *c.* Pain due to pancreatic hemorrhage. It is sudden and extremely severe, attended by collapse. 4. Pain in this situation may be due to aneurism of the aorta, or of the cœliac axis. It is constant, and of a boring character, and may be associated with pain shooting along the course of the lumbar nerves. The physical signs of aneurism are present. 5. Pain in this region may also be due to hepatic colic. 6. It may be due to disease of the vertebræ. We should look for the sixth or seventh dorsal vertebra to be affected, hence higher up posteriorly than the area affected in front would indicate. Finally, 7. Affections of the stomach. Of these we have: *a.* Gastralgia in all its forms. (See Gastric Neuroses.) *b.* Acute and chronic gastritis. *c.* Gastric ulcer. *d.* Carcinoma of the stomach. To the first class belongs a peculiar pain which occurs in locomotor ataxia, which on account of its sudden onset, with alarming vomiting, and its frequent repetition, is known as a gastric crisis.

In diseases of the *stomach* pain is a very common symptom. It is of all degrees, from a mere sense of discomfort or uneasiness to agony. In *atonic dyspepsia* there may be no local gastric symptoms except a feeling of weight and fulness. In *nervous dyspepsia* there is usually uneasiness or discomfort after eating, and in *gastralgia* the pain is characteristic. It usually comes on while the stomach is empty, and frequently recurs daily at the same hour. At first the pain is slight and easily borne, but it gradually increases in severity. It is common for the pain after once reaching its height to subside, and then recur in the same way. Each succeeding paroxysm is worse than the preceding one, until a climax of agony is reached. In character the pain is gnawing and cramp-like, doubling the patient up, and after subsiding, leaving him moist with cold sweat and in partial collapse.

In catarrhal dyspepsia there is pain and uneasiness in the stomach after eating, with tenderness on pressure. If flatulence coexists there will be temporary relief to the discomfort upon the eructation of gas.

In *ulcer* there is a more or less constant feeling of soreness in the epigastrium. After the taking of food the dull pain is aggravated and becomes sharply localized. Frequently also there is pain in the back at the same point a little to the left of the spine and between the mid-scapular region and the lumbar vertebræ. The pain usually occurs sooner after taking food than in the case of cancer, and is more frequently relieved by vomiting. Attacks of gastralgia are not rare, and the pain may shoot down the arm.

In gastric *cancer* pain may be wholly absent throughout the entire

course of the disease, but, as a rule, pain is more continuous than in ulcer, less severe, not so sharply localized, does not come on so soon after taking food, and is not relieved to the same degree by vomiting. Paroxysms of gastralgia are not so common.

In *acute gastritis* the pain and its character vary with the intensity of the inflammation. If due to the irritation of some toxic agent which has been swallowed, the pain is severe and burning; if the result of imprudences in eating and drinking the pain is of a dull sickening character. In either case there is more or less tenderness on pressure. Sometimes in mild cases of catarrhal gastritis firm pressure from a broad surface affords at least temporary relief to the distress.

Pain in the stomach is considered with reference to the taking of food. When it comes on soon after food it is usually due to organic disease of the stomach, as ulcer or carcinoma; but it may be due to neurasthenia. It must not be confounded with the pain that occurs from two to four hours after meals, and is caused by intestinal indigestion or some pancreatic affection.

Alterations of the Appetite. Loss of appetite or anorexia may be due to a number of diseases. It is present in all forms of organic disease of the stomach. It may or may not be present in gastric neuroses. Everyone is familiar with the loss of appetite due to nervous impressions, as emotions, anxiety, or mental care. It is of frequent occurrence in disorders remote from the stomach which modify the condition of the organ reflexly. In the section on Vomiting will be found statements showing the influence of central disease and disease of distant organs upon the stomach whereby vomiting is induced. Through the same channels and through the same mechanism, and hence by the same group of causes, loss of appetite may be produced. Loss of appetite is a constant accompaniment of the moderate gastritis which attends all fevers. Reference cannot well be made to all the conditions which induce this symptom. The writer has been particularly impressed with the importance of determining the presence or absence of suppuration in some portion of the body in all cases in which there is loss of appetite or disgust for food.

Boulimia, or excessive appetite, sometimes occurs. It is popularly thought to be due to worms in children. It is a common symptom in diabetes, and is said to be present in disease of the mesenteric glands. It occurs also in gastric neuroses. Perversion of the appetite, in which all sorts of substances are swallowed greedily, occurs in hysteria, dementia, and pregnancy. It is known as pica.

Regurgitation of gases or food matter is a frequent symptom of gastric disorder. It is also known as belching or eructation. It may be limited to the discharge of gas, although sometimes imperfectly digested food also rises. (See Rumination.)

Regurgitation of the gastric juice alone causes an unpleasant taste, and the fluid is hot and acrid. The juice is usually brought up in the belching of gas.

Pyrosis, or water-brash, is a common symptom in some forms of dyspepsia. It may occur in the morning when the stomach is empty, at which time large amounts of fluid are ejected. The fluid is thin

and watery, sometimes acid, sometimes tasteless. In other cases the fluid is slightly alkaline. The fluid is ejected without vomiting. Sometimes the discharge begins immediately after eating. The late Dr. Chambers thought that the fluid was saliva which was swallowed and retained in the lower part of the œsophagus by a spasm of the cardiac orifice. When a sufficient amount is collected it gushes back into the mouth. Pavy and Handfield Jones believe that the fluid is secreted by the stomach, while, on the other hand, Roberts, who found the liquid to possess diastatic power, believes it to be due to saliva.

Palpitation. Increased action of the heart is a common symptom of indigestion due to flatulency or an overloaded stomach.

Hiccough, or singultus, is a spasm of the diaphragm. The contractions take place at more or less regular intervals, attended with a peculiar clicking sound. This sound is due to the sudden passage of air through the glottis. Hiccough may be a serious symptom. It may last but a few minutes or continue for several days. It causes extreme exhaustion. Its occurrence in chronic disease is of bad prognostic omen.

Constipation. This symptom will be discussed in the chapter on Intestinal Diseases. It is present with gastric dilatation. In pyloric stenosis it is always present.

Diarrhœa. The digestion is impaired and peristalsis is in excess. Lienteric diarrhœa is an accompaniment of a gastric motor neurosis. In gastrectasia the fermentative products set up gastro-intestinal catarrh which induces diarrhœa.

Acute Gastritis.

The simple variety of acute gastritis varies in accordance with the cause, from a slight attack of vomiting after indiscretion in diet, with ordinary symptoms of indigestion, to the more severe forms ushered in by chill and attended by fever.

In the mild forms there is a sense of fulness and discomfort in the epigastrium, attended with nausea. The appetite is lost and there may be disgust for food, and saliva is increased. There is undue acidity. On examination the epigastrium is found to be tender. With the onset of the attack there are giddiness, flashes of light before the eyes, frontal headache and some prostration. The pulse is increased in frequency. When the nausea is most pronounced the face is pale and the extremities cold. Vomiting then occurs, the matter rejected consisting of ingesta only slightly changed, with mucus and watery fluid. It is very bitter. It is often colored green from bile-pigment. Another attack of vomiting may be sufficient to give relief, or it may be repeated for twenty-four to forty-eight hours every hour or two. After the stomach is relieved of the food, mucus and bile alone are vomited.

Examination of Stomach Contents. The reaction of the vomited matter is neutral or faintly acid. No free hydrochloric acid is present, but later lactic and fatty acids are found. Pepsin is lessened.

Twelve to twenty-four hours after the gastric symptoms begin, the intestinal symptoms may arise. Borborygmi and colicky pains are complained of, followed by diarrhœa with some tenesmus.

Herpes labialis may occur, and some writers speak of a peculiar odor which is exhaled from the skin. The more severe cases are ushered in with chill followed by fever. The local symptoms are much aggravated. The tongue is furred, the breath is foul. The vomiting is frequent and severe. The skin is livid and the pulse becomes rapid. In the acute cases attended by fever it may be mistaken for meningitis, peritonitis, or hepatitis. The same gastric symptoms may usher in an attack of pneumonia. The possibilities of a mistake are to be borne in mind, and in all cases of vomiting with fever due regard must be paid to the possibility of the gastric symptoms being symptomatic only. It must be borne in mind that the same group of symptoms due to gastritis accompany the exanthematous diseases, diphtheria and dysentery, pyæmia and puerperal fever. They may be of reflex origin, or due to the action of fever, poison, or ptomaïnes on the stomach. Ewald calls it sympathetic gastritis in which the symptoms are the same as in the simple variety, masked, however, by the primary disease. Sometimes, however, as in the eruptive fevers, attention is directed to the state of the stomach to the exclusion of other conditions. And often to the surprise of the student an eruption or inflammation ensues, which indicates the true nature of the case.

In cases of gastritis, therefore, endeavor to find a local cause for the symptoms. If there is no history of indiscretion in diet, of exposure, of exhaustion, or mental shock, on account of which digestion might be arrested, then inquire for a history of exposure to contagious diseases and look for the earlier evidences of exanthemata. If the pursuit for the cause is still unsatisfactory examine the condition of each individual organ, particularly bearing in mind meningitis, pneumonia, peritonitis, and nephritis.

Phlegmonous gastritis is a very rare affection, in which the inflammation is seated in the submucosa and leads to perforation. The onset is sudden. The local symptoms are intense pain in the epigastrium, with a burning sensation. There is great acidity, dry tongue, and absolute anorexia. The fever is high and characterized by delirium. Chills usually accompany it. The pulse is small, rapid, and irregular. The matters vomited are first mucus, then pus. The patient is extremely restless and anxious, with delirium, and early passes into coma. Death takes place from collapse. It is impossible to make an absolute diagnosis, as local peritonitis, or abscess of the liver are characterized by the same symptoms. In *abscess* a tumor may form in the epigastrium. It may occur idiopathically, but it frequently occurs in septicæmia, and follows trauma.

Toxic gastritis is allied to the former in the severity of general symptoms. It is the result of the swallowing of irritating poisons, of which phosphorus, arsenic, bichloride of mercury and caustic alkalies are the most common. It is attended by inflammation in the mouth, œsophagus and stomach. There is salivation and dysphagia. There is constant vomiting of blood, often with shreds of mucous membrane. The patient is restless, convulsions may occur, collapse rapidly develops. In mild cases, in which the local effects of the corrosive substance may have been mitigated by proper antidotes, sloughs separate, followed by

ulceration, which, when healing, result in deformity or stenosis of the canals.

Some of the poisons are attended by other symptoms peculiar to the special poison. Thus, with arsenic, there are choleraic symptoms; in phosphorus poison the symptoms come on late after its ingestion, and are attended by jaundice and symptoms of acute yellow atrophy.

Mycotic and *diphtheritic gastritis* occur secondarily to typhoid fever, pneumonia, pyæmia, smallpox and sometimes diphtheria. The mucous membrane may be covered with patches in areas or throughout its whole extent.

Some special micro-organisms irritate the gastric mucosa, as the anthrax bacillus; and the sarcinæ and yeast fungi, in cancer and dilatation of the stomach. Rarely tuberculous inflammation with ulceration takes place, and other micro-organisms have been described, as by Klebs. This observer found the bacillus gastricus with numerous spores in the tubules. A gastritis was set up.

The mucous membrane is free from the infection of micro-organisms because of the character of its secretion. The acid gastric juice is antagonistic and causes the death of micro-organisms. On this account, for instance, tuberculosis is rare in the stomach.

Chronic Gastritis.

Causes. 1. Previous attacks of acute gastritis.

2. The local irritation of badly cooked or poorly masticated food, and of alcohol, or other drinks.

3. The local irritation of urea in chronic Bright's disease, or of products of putrefaction in constipation.

4. In anæmia chronic gastritis is of frequent occurrence, and in venous congestions from any cause, but particularly disease of the heart or diseases which interfere with the portal circulation. It occurs secondarily to diabetes, gout, rheumatism, nephritis, and tuberculosis.

5. It is a constant attendant upon local disease of the stomach, as cancer, dilatation, and ulcer, or of local disturbance of the circulation.

The symptoms are those of chronic indigestion. There is a dry, pasty, or salty taste in the mouth, especially in the morning. The tongue is coated over its entire surface, or at the base leaving red patches. The papillæ of the tongue are always swollen and the edges of the teeth marked. Aphthæ recur frequently. The lips are dry and often chapped. The appetite is poor, but varies from time to time. Although there is no great thirst the patients crave fluids with their meals, and acid drinks are grateful. After eating there is an oppression and distention in the epigastrium, followed soon by belching, which is of frequent occurrence. The gaseous eructations are odorless, or foul, and rancid regurgitatian is frequent, with pyrosis. The acidity is due to fatty and lactic acids and not to hydrochloric acid, as in hypersecretion. Vomiting is invariably present, but occurs irregularly. It is usually preceded by nausea. The most characteristic form is that in which mucus is vomited in the morning on rising. Constipation usually

exists; it may alternate with diarrhœa. There is flatulency and rumbling in the intestines.

General Symptoms. The nervous symptoms are the most pronounced. The mental activity is diminished, there is a feeling of languor, or torpor, especially after eating. Headache is frequent after eating, and the patient may become morose and hypochondriacal. Attacks of vertigo are common. Itching of the skin and coldness of the extremities are not rare. Sleep is deeper and longer than we see it normally, but is disturbed by dreams, and not refreshing. Yawning is frequent. Pharyngitis usually attends the attack, on account of which there is hacking cough and expectoration, or hawking of mucus.

The pulse may be weak and irregular, and at times there is an evening rise of temperature. The urine is scanty, high-colored, and usually loaded with urates.

Three forms are seen: (1) *simple chronic gastritis;* (2) *chronic mucous gastritis;* chronic catarrh of the stomach is applied to both conditions. If the condition lasts for a long period of time it results in (3) *atony*, with dilatation of the stomach, or with atrophy. *Atrophy*, or *atrophic gastritis*, is secondary to the chronic form, or to stenosis of the œsophagus, or cancer. The symptoms are those of pernicious anæmia. *Cirrhosis* of the stomach is also a sequence of gastritis. It is rare, and the symptoms are not characteristic of a special lesion. They are those of the primary disease.

Examination of the Stomach Contents. In *simple gastritis* the stomach, after digestion is completed, contains a small amount of slimy fluid. Hydrochloric acid is diminished in quantity after a test breakfast; lactic acid and the fatty acids are present, as previously noted. Pepsin and the milk-curdling ferment are absent or diminished. In *mucous gastritis* there is subacidity. It differs from the simple form in the excess of mucus only. In *atrophy* the hydrochloric acid and pepsin are diminished, or completely absent, after the test breakfast. The fasting stomach is empty. There are no fermentation acids. Atrophy must be distinguished from *cancer* and *subacid neuroses*. The latter occur in younger individuals than those subject to atrophy. A bloody tinge in the stomach contents, or hemorrhage, may be the only distinguishing mark of cancer. Often it is impossible to make a diagnosis.

The diagnostic features of chronic gastritis are: first, long duration; second, persistence of local symptoms; third, recurrence of local symptoms after food, increased by stimulants, or stimulating food; fourth, pain is moderate; fifth, cachexia is absent; sixth, tumor is absent; seventh, hemorrhage is rare; eighth, vomiting may or may not be present, and hydrochloric acid is variable; ninth, flatulency is almost always present. Finally, the cause is usually definite.

Cancer of the Stomach.

The clinical symptoms are varied. It may occur without any symptoms whatever, and be discovered after death from other causes. On the other hand, general marasmus and cachexia may be present alone without local symptoms. In some cases the gastric symptoms are

slight and obscured by the symptoms of secondary growth in the liver or peritoneum.

Typical cases are those which occur late in life, with symptoms of chronic gastritis. These symptoms may continue for months before anything further is observed. Gradually the uneasiness and discomfort after eating increases to actual pain. Loss of appetite is marked, and in spite of careful treatment there is loss of flesh and strength. The usual vomiting of chronic gastritis gradually becomes more frequent. The general appearances of the vomitus are at first like those of chronic gastritis. Soon it becomes streaked with blood, or a moderately large hemorrhage may take place. The vomited matter is dark in color, like coffee-grounds in appearance. The relation of vomiting to the time of taking meals depends upon the seat of the disease. If at the cardiac end of the stomach, the vomiting may take place at once. If in the greater curvature, within twenty minutes or one hour and a half after taking food. When at the pyloric orifice, the vomiting is delayed several hours after food is taken. As the disease advances and obstruction becomes complete at the cardiac orifice food is immediately regurgitated, unless secondary dilatation of the œsophagus takes place. When there is gastric dilatation the vomiting may take place at longer intervals and be characteristic of the vomitus of dilatation. Constipation is the rule.

Tumor. After the symptoms of chronic gastritis have continued for some time without relief a tumor may be detected, depending upon its position and size (see page 478). If the disease is situated at the cardiac orifice of the stomach it is often impossible to detect the growth. If at the pyloric orifice, the tumor is found to the right of the median line above the umbilicus, but may be lowered by the weight of the stomach and felt at the umbilicus. When dilatation follows pyloric tumor it may be lowered still further, as in a case of the writer's, in which it was found two inches below and to the right of the umbilicus. In tumor of the greater curvature the mass is detected below the margin of the ribs on the left side, and may be as low down as the umbilicus. If the greater curvature is involved the organ usually atrophies, and hence the physical signs indicating the lower border of the stomach are higher up than in health.

Symptoms due to Metastasis. The *liver* is the most frequent seat of secondary growths. The organ enlarges and its surface is covered over with nodules. Jaundice rarely occurs. The enlarged liver may cover the stomach and hide the local mass. The *inguinal* glands enlarge, and at times there is enlargement of the *supra-clavicular glands.*

The general symptoms are those of *emaciation* and *cachexia.* The *anæmia* becomes profound. The emaciation is extreme, and in some cases may be out of proportion to the local symptoms. If *fever* occurs in the course of the disease it is usually due to secondary accidents, as suppuration in a tumor, or perforation, with septic peritonitis. The usual course of the temperature is normal until the later stages, when it is subnormal.

The symptoms of *cachexia* are those of emaciation and anæmia. The pallor of the face is striking, and often is of a yellowish and straw-

colored hue. It must not be confounded with jaundice, and examination of the conjunctiva is usually sufficient to distinguish the two. The skin is flabby and the subcutaneous fat is entirely lost; the emaciation is not as marked as in cancer of the œsophagus, except when there is complete cardiac stricture. The nutrition of the skin suffers, boils are common, and ulcers may occur. Subcutaneous hemorrhages are seen in the terminal stages on the backs of the hands, on the dorsum of the feet, on the legs and arms. There is slight œdema of the ankles. General atrophy of the internal organs takes place, so that the heart becomes small; it loses in strength, the patient becomes weaker and weaker, the pulse rapid and feeble.

Examination of the Stomach Contents. Hydrochloric acid is absent in nearly all the cases. For an accurate diagnosis repeated examinations must be made. Other general and local conditions, as fevers on the one hand or dilatation on the other, are attended by absence of hydrochloric acid at times. In carcinoma it is the persistence of the absence which is diagnostic. Pepsin and the milk-curdling ferment are not changed. *Urine.* Indican in increased amount, acetone and diacetic acids, may be present in the urine; otherwise there is no change.

Diagnosis. In the diagnosis of gastric cancer the following must be borne in mind: 1. The age of the patient. 2. The occurrence of causeless dyspepsia without relief. 3. Rapid loss of flesh and strength, with cachexia. 4. The occurrence of pain in the epigastrium, continuous, increased by food, but not relieved by vomiting, as in ulcer, and not distinctly localized. 5. Tumor—hard, circumscribed, followed by the physical signs of dilatation, if in the pylorus. 6. Vomiting is necessarily associated with the taking of food, in which fragments of cancer may be found; blood-cells are common, as detected on microscopical examination, or with Gmelin's test. 7. *Examination of stomach contents.* (*a*) Except in dilatation, the fasting stomach is empty; (*b*) hydrochloric acid usually absent; (*c*) delayed absorption is present, indicated by the motor tests. 8. *Hemorrhage.* In small amounts, usually characteristic, coffee-ground appearance. 9. Metastases—above the left clavicle; in the liver; in the inguinal glands; rarely in the lungs and peritoneum. 10. Eichhorst speaks of persistent itching of the skin and insomnia as characteristic symptoms. 11. Finally, the comparatively short duration of the case. Rarely does it extend over a period of two years.

The Significance of the Tumor. If a tumor is present it is necessary to exclude tumors in the same situation from other causes. This is sometimes difficult. Indeed, as far as the location and physical characters are concerned, often impossible. The most pronounced diagnostic feature of tumor of the pylorus is the occurrence of secondary dilatation. For a differential diagnosis of tumors in this region, see page 478.

DIFFERENTIAL DIAGNOSIS OF GASTRIC CANCER, GASTRIC ULCER, AND CHRONIC GASTRITIS. (WELCH.)

GASTRIC CANCER.	GASTRIC ULCER.	CHRONIC CATARRHAL GASTRITIS.
1. Tumor is present in three-fourths of the cases.	Tumor rare.	No tumor.
2. Rare under forty years of age.	May occur at any age after childhood. Over one-half of the cases under forty years of age.	May occur at any age.
3. Average duration about one year, rarely over two years.	Duration indefinite; may be for several years.	Duration indefinite.
4. Gastric hemorrhage frequent, but rarely profuse; most common in the cachectic stage.	Gastric hemorrhage less frequent than in cancer, but oftener profuse; not uncommon when the general health is but little impaired.	Gastric hemorrhage rare.
5. Vomiting often has the peculiarities of that of dilatation of the stomach.	Vomiting rarely referable to dilatation of the stomach, and then only in a late stage of the disease.	Vomiting may or may not be present.
6. Free hydrochloric acid usually absent from the gastric contents in cancerous dilatation of the stomach.	Free hydrochloric acid usually present in the gastric contents.	Free hydrochloric acid may be present or absent.
7. Cancerous fragments may be found in the washings from the stomach or in the vomit (rare).	Absent.	Absent.
8. Secondary cancers may be recognized in the liver, the peritoneum, the lymphatic glands, and rarely in other parts of the body.	Absent.	Absent.
9. Loss of flesh and strength and development of cachexia usually more marked and more rapid than in ulcer or in gastritis, and less explicable by the gastric symptoms.	Cachectic appearance usually less marked and of later occurrence than in cancer, and more manifestly dependent upon the gastric disorders.	When uncomplicated, usually no appearance of cachexia.
10. Epigastric pain is often more continuous, less dependent upon taking food, less relieved by vomiting, and less localized than in ulcer.	Pain is often paroxysmal, more influenced by taking food, oftener relieved by vomiting, and more sharply localized than in cancer.	The pain or distress induced by taking food is usually less severe than in cancer or ulcer. Fixed points of tenderness usually absent.
11. Causation not known.	Causation not known.	Often referable to some known cause, such as abuse of alcohol, gormandizing, and certain diseases, as phthisis, Bright's disease, cirrhosis of the liver, etc.
12. No improvement, or only temporary improvement, in the course of the disease.	Sometimes a history of one or more previous similar attacks. The course may be irregular and intermittent. Usually marked improvement by regulation of diet.	May be a history of previous similar attacks. More amenable to regulation of diet than is cancer.

Ulcer of the Stomach.

Simple round ulcer of the stomach may occur at any age, but is most common in young anæmic women. It may be the result of an erosion of hemorrhagic infarcts by the gastric juice. Stockton believes it to be a neuropathic change.

THE SYMPTOMS. The symptoms are variable. The cases have been divided by Welch into four classes: (1) Those in which there are

no symptoms whatever, the ulcer having been found after death from other diseases; (2) no symptoms until the sudden occurrence of hemorrhage, or perforation; (3) the symptoms of chronic gastritis or gastralgia only. The symptoms of ulcer may develop suddenly; (4) typical cases. *Pain, hemorrhage,* and *vomiting* are the characteristic symptoms.

Pain. The pain is localized; it is usually confined to a small area in the epigastrium. It may be seated behind the cartilages of the sixth and seventh ribs, or may be complained of in the back, between the eighth and ninth dorsal vertebræ, extending as low down as the first and second lumbar. It is of a burning or gnawing character when seated in the epigastrium, and is said to be gnawing when seated in the back. It is increased by food, and comes on in from two to ten minutes after the ingestion of food. It is relieved by vomiting, or after the act of digestion is completed; but a persistent, dull pain or a feeling of soreness remains. In addition to the ordinary pains, there may be attacks of gastralgia. The pain is increased by pressure. It may be modified by the position of the patient. It may be relieved by lying on the back when the ulcer is in the anterior wall; or relieved by lying on the abdomen when in the posterior wall.

Vomiting. Vomiting occurs shortly after the ingestion of food. It is not attended by retching. The vomited matter may contain blood. The vomited matter and the contents of the stomach contain hydrochloric acid, which may be in excess. Eichhorst thinks it is always in excess.

Hemorrhage. Blood in the vomitus gives it a brown or reddish color. It may be detected by the usual methods. Hemorrhage may occur, however, independently of the act of vomiting. It varies in amount from half a pint to a quart. It may be so severe as to cause collapse. Sometimes, instead of the profuse hemorrhage of dark blood, it may gradually ooze from the ulcer and collect in the stomach before being vomited. It is then altered by the acid gastric juice. Sometimes the blood is not vomited, but passed by stool, which is tarry. Tarry stools also follow the vomiting of blood. In the course of ulcer a hemorrhage may be so severe that death takes place before vomiting occurs. The stomach is then found filled with blood.

The stomach bougie should not be used, and the nature of the contents must be determined by an examination of the vomited matter.

The General Symptoms. If the cases are of long standing, the face is anxious and the lines are sharpened. If there is much hemorrhage, anæmia ensues. There is not much wasting, and there is no fever. Chronic dyspepsia and constipation may attend it during the intervals in which the severe symptoms are in abeyance. The period of abeyance varies and the symptoms may come on without cause, as in gastric crises, during which time the vomiting may persist for two or three days. I saw a young girl of twenty with most severe gastric hemorrhage and classical symptoms of ulcer. With careful treatment she improved. After marriage she remained well until pregnancy. During the first periods of this condition vomiting was very extreme; it then subsided, whereupon, without warning, gastric crisis took place. The vomiting of blood continued for many days, and the symptoms of

gastric ulcer remained for a month. One of the characteristic features of the disease is the occurrence of symptoms which disappear, and after a long period of abeyance recur. A patient under my care, during the last ten years has had three undoubted attacks. It is possible that during each period ulcers healed, to be followed after a time by the occurrence of new ulcers.

Diagnosis. The diagnostic features are: 1. The age. 2. The long duration. 3. The occurrence of emaciation up to a certain point only; most of the patients are under-weight and have a gaunt look, particularly males. 5. The characteristic pain. 6. The vomiting. 7. The hemorrhage. 8. The periods of relief from symptoms. 9. The absence of marked nervous symptoms which attend gastric neuroses. 10. The absence of dilatation of the stomach. 11. The hyperacidity of the gastric juice.

The Accidents of Ulcer of the Stomach. 1. The occurrence of perforation. Sudden severe pain, with collapse. The pain is usually in the epigastrium, but may be in the back as high as the seventh or eighth dorsal vertebræ.

2. Hemorrhage, which may cause death immediately, with either vomiting of blood or with its retention in the stomach.

3. With healing of the ulcer, stenosis at the pyloric orifice may take place with the occurrence of dilatation.

Dilatation of the Stomach (Gastrectasia).

It is caused by obstruction at the pyloric orifice, either from cancer, the cicatrix of an ulcer, or fibrous stricture. It follows atony or degeneration of the walls of the stomach which occurs in the course of chronic gastritis. It may attend paralysis of the stomach. Excessive eating and drinking is the only probable cause independent of organic disease. The dilatation may be *acute.* The term *acute paralytic distention* is also applied to this condition. The cases are extremely rare. There is sudden enlargement of the upper portion of the abdomen, with pressure upon the surrounding structures. The heart is dislocated and its action is very much increased; collapse attends the occurrence, and death takes place from this cause. At first there may be some belching, but the patient is soon unable to remove the gas, and hence occur extreme discomfort, palpitation and dyspnœa.

Chronic dilatation develops slowly. The *symptoms* of it follow the causal disease. They are marked dyspepsia with flatulency, pyrosis and other symptoms of fermentation. If vomiting has attended the causal disease and occurs frequently, its character changes as to frequency of occurrence and the nature of the matter vomited; it now occurs at longer intervals, the amount is excessive, greater than the normal stomach will hold, and is made up of food that is partially digested and fermented and large amounts of mucus. The stomach contents contain sarcinæ, torulæ, and other products of fermentation. Hydrochloric acid is usually absent, but there is a large excess of lactic and fatty acids. With the above symptoms the patient loses flesh and strength and becomes irritable, depressed, and more or less melancholy. The nervous

symptoms of chronic gastritis are also present. In some cases there is excessive thirst because of the small amount of nutrition and fluid absorbed. Palpitation of the heart is common, and dyspnœa may occur on account of the distention. Tetany has been observed in cases of dilatation, especially after lavage.

Physical Examination. The diagnosis is not complete without *physical examination.* On *inspection* the abdomen is large and prominent, and the outline of the stomach can sometimes be seen. Peristaltic movements of the organ are often seen. The movement is from left to right. On *palpation* the peristalsis can be felt, and with one hand on the stomach, tapping with the other, a splashing sound can be detected. A tumor can sometimes be felt in the region of the pylorus, or below the umbilicus. On *percussion,* when the stomach contains gas a tympanitic note is heard. After drinking water, dulness may be detected between gastric and intestinal tympany if the patient stands up. The dull note disappears when he resumes the recumbent posture. The tympany extends high up in the chest on the left side, so that Traube's half-moon space is obliterated. It may extend as high as the fourth interspace on the left side. Cardiac dulness is raised, and the apex of the heart is lifted upward and to the left. In the axillary region the tympany may extend as high as the sixth rib. There is usually atrophy of the spleen, so that unless very careful light percussion is performed the splenic dulness cannot be brought out. The lower limit extends below the transverse umbilical line, and may even extend midway to the pubis. On *auscultation,* succussion can easily be elicited. Sometimes the sound is sizzling as if there were effervescence. Heart sounds may be transmitted clear and metallic over the tympanitic stomach. With auscultatory percussion the border of the stomach can often be defined accurately. Percussion must be commenced far away from the stomach limits and conducted toward it.

Rupture of the Stomach. This may occur in diseased conditions of its walls, or in the healthy stomach from external violence. Pain followed by collapse occurs with almost immediate death.

Functional Disorders of the Stomach.

The Neuroses. Functional disturbances of the stomach are due to impairment of the motor power of the stomach, impairment of the secretory function or of the sensory function. The following table of Ewald, as stated by that distinguished authority, is a classification of the various neuroses midway between the symptomatic and the ætiological:

The Neuroses of the Stomach.

1. Conditions of Irritation.

a. Sensory.	*b. Secretory.*	*c. Motor.*
Hyperæsthesia.	Hyperacidity.	Eructation.
Nausea.	Hypersecretion.	Pyrosis.
Hyperorexia.		Vomiting.
Anorexia *ex* hyperæsthesia.		Colic.
Parorexia.		Tormina ventriculi.
Gastralgia.		

2. Conditions of Depression.

Anæsthesia.	Anacidity.	Atony.
Polyphagia.		Insufficiency of the pylorus and cardia.

3. Mixed Form.

Gastro-intestinal neurasthenia (Dyspepsia nervosa).

4. Reflexes from Other Organs upon the Gastric Nerves.

Reflexes from the brain, eyes, spinal cord, kidneys, liver, sexual organs, and intestines manifest themselves in the forms mentioned in 1 and 2.

It must not be supposed that each of the above-mentioned symptoms occurs separately in an individual, or that functional disturbances may be limited to alterations of the sensory and secretory, or the motor apparatus respectively. They do not occur, as Ewald states, as distinct independent diseases, but usually in groups "either appearing simultaneously, or closely following one another during the course of the malady, passing before us like an ever-changing scene." They may arise directly from disease of the stomach or reflexly from disease of other organs, as the brain, the spinal cord, uterus, kidneys, liver, eyes and nose.

Ætiology. Gastric neuroses are of most frequent occurrence in women, and especially during the years from puberty to the menopause. From the twentieth year onward they are of most frequent occurrence in both sexes because individuals are subjected to the operation of causes which lead to neuroses at this period of life. The gastric neuroses occur in all conditions of patients. They are more likely to occur in those who are poorly nourished or anæmic; although persons who are distinctly robust are liable to have gastric neuroses. While more common in the residents of cities, they may occur in farmers and others accustomed to an open-air life. Although called upon to treat them most frequently among the better classes, nevertheless among the poorer classes a large number of cases are seen. To analyze more closely the predisposing causes we have to study individually all conditions and circumstances in life which lead to wear and tear, as in business or social affairs. In this country, particularly, the causes which Beard and others have forcibly pointed out as factors in the production of neurasthenia are especially prevalent, and are operative in the production of these neuroses.

With regard to men, excess in business or dissipation; in women, excesses in social life or the restraint of home cares, with, unhappily, too often, the irritation of marital relations, are the predisposing factors which lead to the development of this class of cases. Often patients among people of the large cities are subject to the neuroses in the spring after the dissipations of the winter. Behind this excess there is no doubt that a nervous temperament is in the majority responsible for the bringing out of the symptoms, particularly if, combined with this temperament, the patients live in an unhygienic way in regard to exercise, ventilation of their dwelling-places, and drainage, combined with improper diet.

Symptoms. With the gastric neuroses other symptoms of *neurasthenia* are present, and usually the patient may seek advice for these symptoms, such as headaches of various kinds, changes in their mental condition, vertigo, insomnia, neuralgias and paræsthesia of all forms. Intimately

connected with the neurasthenic state is that of hysteria, and therefore in gastric neuroses *hysterical manifestations* are most common. It may be impossible completely to define the border-line between neurasthenia and hysteria, and the gastric symptoms of the former are the gastric symptoms of the latter. While on the one hand, therefore, general neurasthenic symptoms are prominent, in order to accomplish a diagnosis upon which proper lines of treatment can be based, the condition of the individual must be viewed as a whole, and no one symptom or group of symptoms exaggerated in our minds.

Ewald has divided the neuroses into those which arise from *irritation*, and those which arise from *depression*. The first result of irritation is *hyperæsthesia* of the stomach, which is indicated by a feeling of fulness and tension, and of nausea. The sensation is allied to the normal, and is also seen in chronic gastritis, and in the sensations which attend hysteria, meningeal irritation, cerebral tumors, and other diseases of the nervous system. The increased irritability is such that the gentlest irritant excites discomfort or painful sensation. There is a continuous sensation of heat or cold, of gnawing, or pulling, or burning in the organ. The local sensation reflexly influences the psychical life of the patient, so that *hypochondriasis* in some form attends it. The sensations may be relieved by food, to become worse if the stomach is emptied, although in the larger number of cases the trouble is aggravated during digestion. The sensations are likely to be aggravated by fasting a longer period than usual, or by restriction of the diet. Excesses may aggravate them, and on the other hand they are said to follow debilitating states. Some foods, such as shell-fish, crabs and lobsters, or oysters, and strawberries, are likely to increase the peculiar sensations in the epigastrium, exciting mild depression, or burning, or even nausea. The excitation from these foods is usually due to peculiar idiosyncrasies of the individual. On account of the same idiosyncrasies pruritus, erythema and urticaria occur, with headache and some fever.

Deviations from the Sense of Hunger. When hunger is exaggerated it is known as boulimia, or *hyperorexia*. It may be temporary or permanent. When permanent it is obstinate, weakening, and exceedingly unpleasant. It may occur alone or be a symptom of various diseases of the nervous system, manifest disease of the brain, neurasthenia, hysteria, and psychoses. It complicates such disorders as diabetes, and may be of temporary duration in convalescence from acute disease. The disorder accompanies migraine, or hypochondriasis, and exophthalmic goitre. Analogous to it is perversion of the appetite, as seen in pregnancy, in children, and in mental disorders.

Anorexia. Loss of appetite, or repugnance to food. In the first instance, there is simply loss of appetite; in the second, there is repugnance toward food, or nausea at the sight of it. Loss of appetite accompanies dyspepsia in all forms. In the gastric neuroses it occurs spontaneously, or is due to hyperæsthesia of the stomach, and therefore may arise from central or peripheral conditions of irritation. It is commonly seen following central nerve perturbation. The patient is hungry, and sits down to the meal fully prepared to satisfy himself. The first mouthful is at once followed by anorexia, which may almost

amount to nausea. On account of the loss of appetite or repugnance the patient eats less and less of solid food, which results in disturbance of nutrition in a short time, soon affecting the higher centres. Profound mental disturbance may be an exciting cause, so that after the death of a friend, or shock of any kind, the patient is unable to take food. Loss of appetite may be the only manifestation of the gastric neurosis, but because nutrition is so seriously interfered with it results soon in the occurrence of other local or general symptoms. Fenwick points out that the relationship of it to emaciation and enfeeblement are such that grave organic diseases may be simulated. Thus it may be mistaken for phthisis, and general examination alone is sufficient to distinguish it.

Gastralgia. Pain in the stomach occurs in organic disease, as in ulcer or cancer, or forms of gastritis. It also attends the gastric neuroses, and may be the only symptom of this neurasthenic state. Such pain is functional, and is found in anæmic neurotic women. It may, however, occur in all classes. It is characterized by sudden pain in the epigastrium without regularity usually, though at times it may be distinctly periodic. There may not be any definite relationship to the attack of pain and the taking of food, though it is most apt to occur when the stomach is empty. Some classes of food may aggravate it, though, in general, eating usually relieves the pain. If the epigastrium is examined it will be found to be free from tenderness, and indeed often pressure of the broad hand may be a source of relief. The pain is of an agonizing character, sometimes sharply localized, or again diffuse. It may even resemble the girdle sensation. On account of the severity of the pain the patient may be compelled to double himself up to relax the abdominal muscles. The breath is short, and speaking is done in a whisper. The attack is attended by more or less collapse, and the patient may complain of the sensation of impending death. There is pallor of the face, which is distorted with pain, and the brow is covered with perspiration. The pain may radiate along the spinal nerves in close situation to the stomach, and there is often vigorous pulsation of the abdominal aorta.

The attack may last but a few minutes or continue for hours. It sometimes terminates suddenly with vomiting, or is relieved as soon as food is taken. After the attack the patient is exhausted and relaxed, and passes abundance of urine of low specific gravity.

The gastralgias that are due to disease of the central nervous system are often most puzzling. Rosenthal has written exhaustively on this subject. Types of gastralgia of this character are seen in the gastric crises of tabes, first described by Charcot. Recent observers have found that it is due to sclerotic degeneration of the vagus nucleus. The patient is suddenly seized with severe pains, which may begin in the groin and ascend along both sides of the abdomen to the epigastrium, to which point they are fixed. Pain in the shoulders occurs at the same time. The pains are characteristic of lumbar ataxia in their lightning-like rapidity. With the pain the heart's action is increased in rapidity and force. There is no rise in temperature. At the same time there is uninterrupted and painful vomiting, which is attended by nausea and vertigo. The gastric pain may continue uninterruptedly

for two or three days. It belongs to the pre-ataxic period, so called, but is almost sure to continue throughout the whole course of the disease. The nature of the stomach contents bears no relation to the pain; the frequency is variable. The pains may recur at long periods, or as frequently as once a month or once a week. Another special characteristic is the sudden relief that is given without cause.

Neurasthenic Gastralgia. Neurasthenic gastralgia occurs in patients who are suffering from neurasthenia, and is divided by Rosenthal into two forms, the one irritative, the other depressant; these are related by transitional forms. The early symptoms of neurasthenia (*q. v.*), and particularly in the irritative form with painful points in the nape of the neck and between the scapulæ, or often lower down on the vertebræ, with neuralgias and paræsthesia in the upper and lower extremities, are attended by periodical recurring gastralgia. The gastralgia is characterized by a boring sensation which, during the attack, radiates over the lower ribs to the median line. It is accompanied by vasomotor symptoms and symptoms of cerebral anæmia. In the *depressant* form the patient complains of weight and fulness, or a dragging sensation after eating, which is constant instead of paroxysmal. The neuralgic pains are not so marked, motor exhaustion is not so prominent, and the pain in the back is not as intense so in other varieties. In both instances on deep pressure over the region of the nerve plexuses which follow the bloodvessels in the abdomen, there is sharp and unpleasant pain radiating to the epigastrium. Burkart considers these painful points to be present in all cases, while Richter believes that pressure over the stomach and abdomen is not painful. With such pain there is usually increased pulsation of the abdominal aorta, particularly during the time of the paroxysm. In neurasthenic gastralgias there is increased sensitiveness to the electrical current and increased stimulation of the sensory nerves of the trunk, which may also be extended to the limbs.

Neurasthenic gastralgia must be distinguished from the gastralgia of organic disease and the gastralgia of hysteria. The gastralgia of organic disease is recognized by observing the condition of the stomach when fasting and by studying the secretion. In organic disease there is retarded digestion; in gastric neuroses digestion is completed in the normal limit of time, seven hours. Hysterical gastralgias are recognized by the presence of the usual symptoms of hysteria, in which the psychical factors occupy a prominent place, associated with convulsions, paralyses, pupillary inequalities, hemianæsthesia and electrical sensibility. Most characteristic, however, is the alternation of hysterical gastralgia with neuralgia, or neuroses in other organs.

Hyperacidity and Hypersecretion. Hyperacidity is the increase of the normal amount of hydrochloric acid secreted, due to a neurosis of the secretory function. Hyperacidity begins when the amount of acid in the fluid withdrawn from the stomach in the usual way is between 60 and 70 per cent. It must not be forgotten that it is a symptom of gastric ulcer, but it exists as a neurosis independent of any organic lesion of the stomach. It has been observed in nervous diseases, as hysteria and melancholia, and as a reflex symptom in gall-stones and renal calculus.

Hypersecretion occurs in two forms, the *periodical* and the *constant*. The acid is not necessarily increased. The periodical occurs after eating; it does not have direct connection with food. It is seen in neurasthenia or locomotor ataxia. In chronic hypersecretion the gastric juice, which is usually hyperacid, is in excess, so that the fasting stomach may contain large quantities, even to a pint and a half, without food and only slightly tinged by bile. In chronic hypersecretion the digestion of starches is delayed, but that of albuminoids is very prompt. After an abundant meal consisting of meat and starches the meat disappears entirely. Hypersecretion occurs in about half of all the stomach disorders, according to Riegel. It is more common in men than in women. The acid fluid causes the hyperæsthetic conditions previously described in the gastric region. Pain and eructation, heartburn, or gastralgia, vomiting of sour masses, occur with the digestive disturbances of chronic gastritis. The tongue is usually clean and the appetite increased rather than diminished. Acidity is common. As a result, atony of the muscular coat takes place, followed by gastrectasis. The neurosis is then converted into an organic lesion, and the symptoms of dilatation arise.

In order to make a diagnosis the secretions must be secured while fasting. The patients usually improve on albuminous food, which differentiates it from gastralgia and pyrosis of acid fermentation. Alkalies give temporary relief.

Gastroxynsis is a gastric neurosis in which, after mental over-exertion or profound emotional disturbance, there is sudden vomiting, continuing for a considerable time, of acid fluid. It is closely allied to migraine. Nervous *belching* and *eructations* are phenomena of the gastric neuroses of motor origin. They usually occur in hysterical subjects rather than in neurasthenics. In the latter they are associated with other sensations, particularly oppression and tension in the epigastrium. In hysteria they occur alone. There is increase in the contractility of the stomach, the pyloric sphincter contracts powerfully, and the stomach is distended; gas is expelled at the cardiac end of the stomach. They may be due to paralysis of the cardiac end of the stomach rather than contraction of the pyloric end. They occur involuntarily generally. They must not be confounded with the pseudo-hysterical vomiting which Bristowe has described. In this instance the gas is raised from the œsophagus by contraction of the muscles of the neck. Hysterical eructation is very frequently of œsophageal origin. The belching is loud and may occur in paroxysms. The gas is odorless, and hence is distinguished from the gas of dyspepsia and fermentation; it is in all probability the result of the swallowing of air.

Pyrosis, heartburn, is the raising of sour masses from the stomach. The stomach contents are not necessarily hyperacid. If acid, as in the normal gastric juice, or hyperacid, the regurgitation causes severe acrid and burning sensations. It is probably due to heightened contractility of the muscular coat of the stomach with pyloric contraction, which overcomes the weaker cardia.

Pneumoptosi. Excess of gas in the stomach. When the stomach is overdistended, in addition to the tension, the diaphragm is pushed up, pressing on the heart. The patients are seized with severe dyspnœa.

At first inspiration is difficult, and finally both inspiration and expiration become difficult. Palpitation of the heart and pulsation of the peripheral arteries take place. There is fulness of the head and a sensation of impending death. The patient may become unconscious. Relief can only be afforded by belching, when the attack rapidly subsides. Introducing a stomach bougie gives immediate relief.

Nervous Vomiting. (See Subjective Symptoms, and Gastroxynsis.)

Peristaltic Unrest. Characterized by borborygmi and gurgling, which begin immediately after eating, are heard at a considerable distance from the subject, and are a source of great annoyance. It is a common symptom of the gastric neuroses.

Rumination. Rumination is a rare condition in which the patients regurgitate and chew the cud like ruminants.

Conditions of Depression. In conditions of depression *polyphagia*, or the want of a feeling of satiation, which, if gluttons are excluded, is a morbid condition of extreme rarity.

Anacidity of the gastric juice as a neurosis is found in hysterical persons and in neurasthenics. (See Absence of Hydrochloric Acid.)

Relaxation of the Cardiac and Pyloric Ends of the Stomach from Conditions Resembling Paralysis. When the cardiac end is relaxed, eructations and regurgitations occur. If large quantities of the material from the stomach are regurgitated and expectorated, the condition is pathological. It may lead to serious changes in nutrition. It may exist for years without bad results. It must not be confounded with the regurgitation from diverticula of the œsophagus. In the latter regurgitation is produced at will.

Rumination (*Merycismus*). See above.

Atony, or Atonic Dyspepsia. It accompanies gastritis; it also occurs as a primary neurosis. The innervation of the nerve centres regulating peristalsis is disordered. The primary disorder may be local or central. The movement of the chyme is tardy or insufficient. Atony should be applied to the disease of the motor function only, or, as Rosenbach states it, to insufficiency of the stomach. The symptoms develop gradually. At first occurs oppression during digestion, with swelling and fulness of the stomach. There is mental and physical torpor during the time of the digestive act. The symptoms become aggravated, and eructations occur, vomiting begins, and gradually the fermentative symptoms become most pronounced. At this period it is putrid, or fermentative dyspepsia. By the usual tests the motor power of the stomach is found to be diminished. The secretions are also scanty.

Nervous Dyspepsia. According to Ewald this is the true gastric neurasthenia, which combines all forms of gastric neuroses. The clinical picture is made up of a combination of all the neurosal symptoms mentioned. Leube considers nervous dyspepsia a group of symptoms of a cerebral nature due to abnormal irritability of the sensory nerves of the stomach during the normal digestive processes, the symptoms of which are hyperæsthesia and nausea, hyperorexia, anorexia, parorexia, and gastralgia. Leube thinks the true peptic activity of the stomach is unchanged. While the anatomical or physiological explanation of the condition is difficult, the clinical symptoms are those

of irritation or paralysis, the manifestations of which are intermingled, sometimes one and sometimes another being most prominent. (See table, page 513.)

The one characteristic feature is that the symptoms are mild. With severe forms of gastralgia nervous vomiting and boulimia do not occur. Symptoms of intestinal indigestion are usually associated in a mild degree. Constipation is of the most common occurrence, although in some cases there is diarrhœa. In other cases the intestinal indigestion is much aggravated with mild gastric disturbances and anorexia, repugnance toward taking food, furred tongue and mild nausea; and there are constipation and colicky pain, either diffuse or in separate painful spots. The abdomen is distended and tympanitic, sometimes to a marked degree. It is called *flatulent dyspepsia*. Along with the gastric and intestinal symptoms the general nervous symptoms to which the term neurasthenia is applied are present. These nervous manifestations sometimes precede the local gastric symptoms, but as the latter develop the former become more aggravated. The dyspeptic conditions, as Ewald puts it, are on a neurotic basis, or such as may occur as reflex neuroses in chlorosis, menstrual disorders, uterine and ovarian disease, and intense psychical excitement. Where pathological and anatomical changes are lacking, as far as is known great alterations in the chemical functions are absent. An indigestion of short duration, a mild catarrh, recurring hyperæmia, have been the primary causes of nervous symptoms in the digestive organs.

Diagnosis. There are no characteristic symptoms, and the student must bear in mind that it may be necessary to make several examinations and listen to the story of the subjective symptoms frequently before a complete conclusion can be arrived at. This is all the more necessary because of the frequency of organic lesions and neurasthenic conditions being present at the same time. The course of the disease must be observed for a long time, all possible causal factors investigated and all the general signs of neurasthenia carefully considered. In addition it may be necessary to use therapeutic tests. If the possible organic diseases are not relieved by such measures there must be a deeper basis for the gastric symptoms. Just as in neurasthenia and in neurasthenic states elsewhere, the individual must be considered as to peculiarities, idiosyncrasies, and all his relations in life, in connection with the general and local symptoms of the neurasthenic state. Great stress must be placed upon the study of individual symptoms, their mutual relationship and their changeable occurrence. In gastric neurasthenia, gastralgia is more diffuse than the pain of ulcer or cancer of the stomach. It is not so much dependent upon food as either of the others, particularly ulceration. In gastric neurasthenia vomiting is rare. The vomitus is composed of mucus mixed with bile and food in various stages of digestion. It is never bloody nor does it contain decomposed masses. Hysterical vomiting occurs with ease and regularity compared with the vomiting of neurasthenia. The vomiting in neurasthenia is bitter, due to the presence of peptones. In gastric neurasthenia the stools are changeable in character. They do not contain undigested remnants of food, or mucus, or blood. The form of the fæces is variable.

Differential Diagnosis. Neoplasms, ulcers, strictures, dilatations are distinguished by physical signs or characteristic symptoms. In gastric neurasthenia the stomach should be empty seven hours after taking a meal. The results of the chemical examination are not sufficiently definite for diagnostic purposes, for at times the same chemical changes are present as in ulcer, carcinoma, and chronic catarrh. The diagnosis must be based largely, as previously intimated, upon prolonged observation and a carefully taken history, and upon the general condition of the patient. The cases must not be mistaken for costal neuralgia, although it is not usually easy to be led astray. Reflex gastric neuroses are seen, as indigestion, gastralgia, or vomiting. The types are interchangeable, although vomiting occurs in the more acute reflexes, indigestion in the more chronic. The cerebral disorders which give rise to vomiting are meningitis, abscess, and tumor. The vomiting may be transitory, or may be persistent. There is usually hypersecretion of the gastric juice. The vomiting may usher in the disease or develop during its course. If vomiting is of long-standing its possibly reflex origin should always be investigated. (See Vomiting, page 497.)

Gastralgia is sometimes a reflex from lesions in the cervical and dorsal portions of the cord; not only in the posterior columns, but also in disseminated sclerosis. Vomiting occurs, and the attack is known as a *gastric crisis.*

Chronic dyspepsia is a frequent reflex disorder on account of diseases of the sexual organs, as amenorrhœa and dysmenorrhœa, in the climacteric period, and in chronic inflammations of the uterus. In malpositions and tumors, and in pelvic exudations with traction, in ulcers, in ovarian tumors, the so-called dyspepsia uterina of Kisch is common.

The Stomach in Other Diseases. Diseases of the stomach may frequently mask other diseases; in other words, patients will complain of gastric symptoms which, however, are concomitant phenomena, behind which there are graver conditions. Thus, in disease of the kidney, in phthisis, in chronic gastritis, in emphysema, in valvular disease of the heart, catarrh of the mucous membrane of the stomach is of frequent occurrence, depending upon the primary disease. In *tuberculosis* the local gastric symptoms often seem to be the more prominent features. Thus, in the earlier stages of phthisis, loss of appetite and vomiting are of constant occurrence. The dyspeptic symptoms in a large number of cases precede the pulmonary symptoms and may be so pronounced as to mask entirely the symptoms of the latter disease. The patients are usually delicate and anæmic; they complain of loss of appetite and mild indigestion; there is some regurgitation of food; they are feeble and languid; they are treated for chronic catarrhal gastritis, but do not improve. On examination of the lungs the physician is surprised to find a small area of consolidation, and upon inquiry will find subjective symptoms of tuberculosis to have been present for a considerable time. Every practitioner is familiar with the scores of patients with phthisis, which may even be advanced, who believe that their symptoms are entirely due to disorder of the stomach. In addition to the early catarrh that precedes tuberculosis, other gastric symptoms may occur. The well-known association of ulcer in phthisis is familiar,

although there is probably no causal relation, because both occur at the same time in life, yet the gastric symptoms may prevent investigation into those of pulmonary origin. In *anæmia* and *chlorosis* the changes in the digestive tract are common. On account of the general blood condition the functions of the stomach are impaired. Here, too, we frequently have the association of ulcer with the general condition. Danger of overlooking either is not so great as in tuberculosis.

Valvular Affections of the Heart. Chronic catarrh of the stomach is liable to occur on account of venous congestion; the symptoms may point to the gastric condition alone. In all cases of chronic gastric catarrh it is necessary to examine carefully into the condition of the heart. Over and over again patients apply for treatment on account not of cardiac symptoms, but because of gastric disorder. They will be treated in vain unless the primary affection is ascertained. Many cases of gastric catarrh have been cured by the use of digitalis. In disease of the *kidneys*, the stomach is frequently involved. Vomiting and other symptoms of gastric indigestion may occur long before dropsy or any objective sign which would lead to a correct diagnosis. The gastric symptoms are due to chronic uræmia. In other conditions of the genito-urinary tract, gastric symptoms also occur. This is particularly noticeable in long-standing retention from chronic obstruction. Renal tumors may cause only disturbances of digestion, while gastric symptoms due to movable kidney are well known. The symptoms in the latter condition arise, first, from mechanical causes, as the pressure of the kidney on the pylorus, and secondly, from the influence on the nervous system.

Disease of the Liver. The intimate relationship of the liver and the stomach is such that when one is the seat of serious functional disturbance the other is likely to be affected. Frequently it is impossible to draw fast lines as to which organ is the primary seat of disorder. In the use of alcohol chronic gastritis is of frequent occurrence, and this intoxicant also causes cirrhosis of the liver. On the other hand cirrhosis of the liver is frequently accompanied by chronic gastritis secondary to a portal congestion.

Diseases of the Central Nervous System. The relationship of disease of the central nervous system to those of the stomach has frequently been adverted to. (See Vomiting.) In sclerosis of the posterior columns of the cord this is more striking than in any other condition. Not only do we have gastralgia and gastric crises, but moderate symptoms of indigestion, with hyperæsthesia and slight gastralgia, may be the first symptoms of lumbar ataxia.

Diabetes. Diabetes may continue in its course for a long period of time, during which the patient is thought to have some stomach trouble, when an examination of the urine reveals the true nature of the case. In *gout* and the *rheumatic diathesis* opinions differ as to the relationship of the stomach to this disorder. Some writers are full of the belief that a specific gouty inflammation of the stomach, due to the uric acid diathesis, is of frequent occurrence, and that one of the prominent manifestations of gout is dyspepsia in all its forms. The French consider gastric disturbances to be frequent expressions of the rheumatic diathesis. The relationship of the two, however, is thus far not fully developed,

although, in these conditions, it is not usual to overlook the presence of either of the diatheses when symptoms of gastric disturbance occur. It is essential to bear in mind that in persons of a rheumatic or gouty diathesis gastric disturbances are as liable to occur as in healthy individuals; their successful management depends upon the recognition of the fundamental diathesis.

Diseases of the Intestines.

The intestine is a canal of varying dimensions, the physiological office of which is to propel material received from the stomach, and to permit of the digestion and absorption of that which is to serve for the nutrition of the body. The canal is richly supplied with bloodvessels and lymphatics. It is made up of mucous membrane, muscle and peritoneum. For the purpose of digestion, fluids are secreted, either from the intestinal glands or large neighboring glands which discharge into the canal.

Diseases which affect the canal impair or cause an abeyance of the physiological offices. As these offices—absorption and digestion—are essential to nutrition, it is not surprising that the body weight and strength are impaired. We know too little about the function of digestion to utilize such knowledge in diagnosis. Intestinal digestion is also dependent upon the healthy performance of the functions of the liver and pancreas. It is difficult to draw fine lines of distinction even in health, and intestinal pathology is closely interwoven with hepatic and pancreatic pathology.

Alterations of the function of the intestine as a canal give rise to distinctive symptoms. Either its movements are too frequent and rapid, causing *diarrhœa*, or too sluggish, causing *constipation*. Obstruction of the canal leads to symptoms common to such a condition (see Morbid Process), modified by the physiological duties and the anatomical structure of the canal.

The morbid processes are hyperæmias, inflammations, degenerations and new growths. The symptoms that attend these processes are not different from the symptoms that attend such processes in similar structures elsewhere. It must not be forgotten that the function of the canal is influenced by each process. On account of the process we may have *pain* and *fever*; on account of impaired function, *pain*, *flatulency*, *diarrhœa* or *constipation*, *change in the character of the stools*, and *impaired nutrition*. Some of the above morbid processes may lead to the mechanical condition, *obstruction*.

The morbid alterations of the intestinal tract are ascertained by data derived by inquiry and by observation. The data derived by inquiry include the subjective symptoms—pain, and discomfort from flatulency. By observation, the general condition of the patient is noted, the presence of tenderness, alterations in the size and shape of the abdomen, and other physical phenomena observed. The fæces are carefully studied, with the object of determining modifications of the function of the bowel, the presence of ingredients due to some morbid process, as serum, blood, pus, or mucus, or of extraneous matter, as

worms or foreign substances. The fæces are studied by the naked eye, by the microscope, and by bacteriological methods.

One symptom may be the chief manifestation of a disease, as pain of lead colic; diarrhœa of several morbid disorders; constipation of others. In the discussion of the special symptoms a consideration of the diseases of which the symptom is the main expression will be taken up.

The long channel is the recipient of material for nutrition, which may contain parasitic forms of animal life or their ova or spores, which enter the body in this manner. They remain in the intestinal tract or wander into other structures. They include animal and vegetable parasites. To the class of parasites belong forms of protozoa, vermes, and fungi. While the canal is open to infection by various micro-organisms, it is the natural habitat of others which may become deleterious agencies when the conditions of the environment of the parasite are changed. Thus the bacillus coli communis is, in man, with normal epithelial structure and normal secretions, an innocuous parasite which, when inflammation sets in, may become nocuous.

The symptoms of the protozoa and fungi, or of their products, the ptomaïnes, are of an infectious or toxic nature. Inflammation is produced locally, while general infective or toxic symptoms occur.

The symptoms of worms, if retained in the intestinal canal, are—

1 Reflex in nature; (2) symptoms due to catarrhal inflammation; (3) symptoms due to the action of the parasite on the blood—anæmia; (4) symptoms due to wandering of the parasite, as in trichinosis. (See Fæces.)

Symptoms of the Tæniæ and Bothriocephali. There may be no symptoms save discharge of the parasite or portions of it by the rectum. In others the symptoms of intestinal dyspepsia or intestinal catarrh are observed. Headache, giddiness, lassitude, and itching at the nose and at the anus are said to be present. The patient becomes hypochondriacal. Convulsive disorders occur. Hysteria, forms of epilepsy, grinding of the teeth at night, and restlessness attend the habitation of the parasite in the intestine. In all convulsive disorders, the possibility of worms as a cause must be remembered.

Symptoms of Ascarides. (1) Gastro-intestinal catarrh; (2) symptoms of obstruction (rare); (3) symptoms due to wandering—as to the hepatic duct or to the stomach, to the vagina; (4) nervous symptoms of reflex origin; (5) the worm or its ova in the fæces.

Symptoms of Oxyuris Vermicularis. (1) Gastro-intestinal dyspepsia or catarrh; (2) itching or heat at the anus, worse in bed; (3) vesical and rectal tenesmus; (4) erythema about the anus; (5) priapism; (6) vulvitis and vaginitis; (7) the worms in the fæces.

The Strongylus. The symptoms are local, with the symptoms of profound anæmia. The discovery of the ova in the fæces distinguishes this form of anæmia from other varieties.

The symptoms due to the presence of the trichina spiralis and filaria will be discussed in appropriate sections. (See Blood and General Diseases.)

The *intestines in other diseases.* The relationship of intestinal disorders to affections of other viscera will be discussed with each symp-

tom. It must not be forgotten that derangement of this tract may have its origin in local causes or in causes remote from the intestinal tract, or in some general condition of the individual. Thus diarrhœa may be due to inflammation which is primarily local, or which may be secondary to infection. Nothing is more common than to see diarrhœa with general infection, as septicæmia. In exophthalmic goitre the diarrhœa is not due to a local cause, but to some, not yet known, nerve disorder. Constipation may be due to central brain disease, to a general condition like diabetes, or be of local origin.

It must be remembered that the diagnosis of an intestinal lesion is never complete without determining its causes. Thus enteritis and ulceration occur in typhoid fever, in cholera, and in other infectious disorders, all of which are to be passed in review in making up a diagnosis. Diarrhœa is a symptom in Bright's disease, and the causal relationship must always be borne in mind.

Intestinal diseases or disorders are not usually confounded with disease of other structures. It is worthy of remark that symptoms of intestinal obstruction are frequently due to peritonitis, the latter condition being overlooked. Tumors of the intestine must be distinguished from tumors of the peritoneum, the stomach, pancreas, and liver, and the uterus and ovaries. The history, the seat and physical character of the tumor, and the associate symptoms, point to the true condition.

Arteries of the Intestine. The intestines are supplied by the mesenteric arteries. Its branches may become the seat of *emboli.* The symptoms are sudden pain, intestinal hemorrhage, and discharge of a portion of intestine. The patients are the subjects of atheroma or heart disease.

The Data Obtained by Inquiry. The Subjective Symptoms.

Pain. *Colic.* Colic is the term applied to paroxysmal pain in the abdomen. It is further characterized by suddenness of onset and by alteration of intestinal function. It attends all forms of inflammation of the intestinal tract. It is applied to a peculiar affection known as lead colic, due to the local effects of lead. Enteralgia should, however, be applied to this form. The term *colic* is also applied to painful affections of the hepatic ducts, pancreatic ducts, the ureters, and the uterus. Intestinal colic is the form at present referred to. In addition to the inflammations of the intestinal tract it may be due to *indigestion* with flatulency. When it occurs suddenly without local cause it is known as enteralgia. It is a nervous affection.

The colic of intestinal indigestion occurs suddenly, or may be preceded by signs of intestinal indigestion. The pain is chiefly in the umbilical region and radiates from that point. It is relieved by moderate pressure or by warmth. The patient is restless and irritable. The face is anxious. The pain causes him to roll about and double up. There is a cold sweat, and the pulse is small and hard. Nausea and vomiting follow the pain, and there are gaseous eructations. The abdomen is distended from gas, and tympanitic on percussion. Prostration or collapse rapidly ensues. The pain may be relieved by the passing

of flatus. With the local pain there is spasm of the muscles of the calves. The cramps are very painful; the muscles become knotted. The hands and feet are also cramped. The pain is said to be due to spasm of the intestine, and is known also as spasmodic colic. It is certainly due to distention or to irritation.

If the intestinal colic is due to *indigestible food* it may have been preceded by an attack of acute indigestion, and the griping pains may have developed at long intervals, with gastric and intestinal flatulency. Vomiting may precede or attend the attack, and diarrhœa follow. If the colic is due to *gas* alone there is great tympanites. If it is due to *fæces* it has been preceded by a history of constipation, and there may be fæcal masses detected in the rectum or along the colon.

Diagnosis. The sudden severe pain, often relieved on the discharge of gas, with gastro-intestinal disorder, tympanites, the occurrence of cramps in the extremities, and the localization of pain to the umbilicus, all point to the true nature of the affection. A history of indiscretion in diet, or exposure, aid in the diagnosis. In colic the pain may come on suddenly or increase gradually from a sense of discomfort or soreness. The pain at its height is described as agonizing, and of a boring or shooting character, abating for a time and then increasing until the patient rolls and twists in agony and breaks out into a cold sweat. The pain may shoot from the seat of greatest intensity to the shoulders, back, chest, or iliac region.

It must be distinguished from *enteralgia.* The latter comes on slowly and lasts for hours or days. The pain is situated around the umbilicus, it is relieved by deep pressure, although the skin may be hyperæsthetic. Sometimes the abdomen is retracted; there are no signs of indigestion, and flatulency and borborygmi are absent.

Lead Colic. If the enteralgia is due to *lead* there is a history of exposure to that metal. The blue line on the gums, with obstinate constipation but no vomiting, and the occurrence of neuritis in other situations, due to saturnine poisoning, point to the true nature of the case.

Hepatic Colic. In hepatic colic the pain is situated in the region of the liver and may radiate to the shoulder or back. It is sometimes fixed in the parasternal line about the cartilages of the sixth and seventh ribs. The attack is attended by vomiting, usually of bilious fluid. It occurs in women most frequently; almost always after forty in both sexes. It may be followed by jaundice. There is local tenderness, and there may be some swelling in the region previously mentioned. The bowels are constipated, and after the attack may contain gall-stones.

Renal Colic. In renal colic pain begins in the kidney and then extends along the ureter. It is always more localized to the right or left of the median line in the abdomen. It is more frequent in the lower portion of either of the upper quadrants, three inches to either side of the median line, depending upon the kidney affected. From this region the point of maximum intensity and of local tenderness moves to the lower quadrant toward the median line in the oblique direction, rarely getting an inch below the transverse umbilical line. The pain then

extends to the region above the pubes and down the thighs. From the first there is increased frequency of micturition. The urine is scanty, high-colored, and may contain blood. With the free micturition relief follows.

Local Peritonitis. Pain connected with the liver, spleen, and kidneys is generally due to involvement of the peritoneal coverings of these organs, and partakes of the character of local peritonitis. It may, however, be due to malignant, ulcerative, or inflammatory disease, and the diagnosis must be made by noting the character of the pain, its intensity, duration, seat, and the other general and local symptoms with which it is associated.

Rectal Pain. Pain in defæcation may be due to piles, internal or external, or to fissure, or may be the result simply of the passage of an unusually large, hard mass. Pain from fissure is most acute and spasmodic, and persists for some time after defæcation. Fibroid stricture of the rectum causes more pressure and straining at stool than real pain. But cancer is apt to be extremely painful.

Uterine Colic. In uterine colic the pain is situated in the pelvis. There is some abnormality of discharge, and a history of uterine disease. Care must be taken not to confound the sudden pain of extra-uterine pregnancy with intestinal colic or other forms of abdominal pain. In *extra-uterine pregnancy* the pain is in the lower quadrants of the abdomen to the right or left of the median line. It is sudden and intense pain attended by more or less collapse. It may be attended by all the symptoms of internal hemorrhage. It may cause vomiting. The history of cessation of menses, of discharge of decidua, or other signs of pregnancy, with the local signs on physical examination, indicate the true nature of the pain.

Pancreatic Pain. In *disease of the pancreas*, either from the passage of calculi (extremely rare) or because of pancreatic hemorrhage, there may be sudden severe pain. The pain is localized to the region below the sternum. It may be severe in the back and extend up the thorax. It occurs in paroxysms, and is attended by great anxiety and collapse.

Gastric Pain. Intestinal colic must be differentiated from pain of gastric ulcer, gastric cancer, and gastralgia. The characteristics of pain in these affections will be discussed subsequently. When perforation occurs in gastric ulcer the pain is usually seated in the epigastrium, but may be complained of in the back as high as the mid-scapular region. It is sudden and severe, preceded by a history of ulcer and attended by collapse. There are no evidences of indigestion. Perforation of the biliary passages is attended by pain in the hepatic region. The pain is sudden and is usually preceded by symptoms due to derangement of the biliary passages, by obstruction of gall-stones. Pronounced collapse follows its occurrence.

Appendicitis. Intestinal colic must not be confounded, although it frequently has been, with the pains that attend appendicitis. This is particularly the case with relapsing appendicitis. In this form only mild fever attends the attack. The patient is seized with severe pain, which may be described as occurring in the lower right quadrant, but is sometimes complained of about the umbilicus. It frequently follows indis-

cretion in diet, and may be attended by vomiting, and is likewise usually relieved by eructation, but not by the passage of gas, a point of great importance in the diagnosis. The attack occurs mostly in young subjects and continues but twelve to twenty-four hours. It may be so severe as to cause collapse. If fever attends it, and there is true appendicitis, the diagnosis is much easier. In the relapsing as well as the true form there is tenderness at McBurney's point. (See Appendicitis.)

Peritonitis. Intestinal colic must not be confounded with *peritonitis*, which follows in all the above conditions, or develops at various points in the abdomen. The purulent peritonitis that succeeds pyosalpinx may be attended by severe pain without much reaction. The pain, however, although complained of about the umbilicus, can be localized by pressure in the lower quadrants and in the pelvis. It may disappear after eight or ten hours, to be followed by a recurrence. The recurrence of pain is usually attended by fever. In the first twenty-four hours the bowels are loose, or at least readily moved. If the peritonitis continues it is impossible to move the bowels often.

Organic Disease of the Bowels. Intestinal colic must not be confounded with organic disease of the bowels on account of which obstruction arises. In these affections there is sudden constipation, and rapid prostration. The vomiting, if present, persists and soon becomes stercoraceous. In *intussusception* the stools are characteristic. Strangulation or ileus is associated with the presence or history of previous peritonitis or hernia. In the latter there may be signs at the hernial points. In the obstruction from external pressure the presence of tumors has been known previously or can be recognized. In fæcal obstruction, or the obstruction by gall-stones, the local signs may be pronounced, and the pain is usually in the ileo-cæcal region. The above-mentioned source of pain, which may be confounded with intestinal colic, usually occurs suddenly. The affection is acute. Pain that extends over a long period of time, that is not due to an acute process, or attended by severe acute symptoms, has been considered elsewhere (see Abdomen).

Abdominal Rheumatism and Neuralgia. Intestinal colic may be mistaken for *rheumatism* of the abdominal walls. In the latter there may be a history of exposure. The muscles are extremely tender. There are no gastro-intestinal symptoms, the urine is loaded with uric acid and urates, and there may be muscular pain in other situations, or a pronounced history of previous attacks of rheumatism. In lumbo-abdominal neuralgia the pain may simulate intestinal colic. Pressure-points where the respective nerves exit through the fascia are detected. Just here may be considered the pain about the navel, which occurs in paroxysms, due to disease of the vertebræ. There may be caries from tuberculous disease, or from pressure of an aneurism. Examination of the vertebræ may determine its nature.

Fever. The occurrence of fever points to inflammation in some portion of the gastro-intestinal tract or the abdomen in the diagnosis of intestinal colic ; moreover, in the former the pain is constant, but localized and aggravated by pressure. The skin is hot and dry.

Diarrhœa. Diarrhœa is a symptom of disorder of the intestine which in turn is itself the cause of symptoms, just as jaundice, a

symptom of hepatic disorder, is the cause of various symptoms. In diarrhœa there is increased frequency of the movements of the bowels. This is due to increased peristalsis of the intestine, which occurs from a number of causes. Not all increased peristalsis results in diarrhœa. (*A*) Increased peristalsis may be due to some impression upon the nervous mechanism of the intestine. This may explain the diarrhœa of emotion, or that which occurs from other psychical influences. (*B*) On the other hand, in the larger number of cases the diarrhœa is due to catarrhal inflammation of the intestinal tract. The causes of the catarrhal inflammation are many, and have been divided into primary and secondary causes. *Primary* catarrh is due to the direct influence of causal factors upon the mucous membrane. (1) It is seen after cold or exposure; (2) it occurs from the direct action of an irritant, as undigested food, and (3) from the action of irritants, as of bacteria or the products of bacteria. Catarrhal inflammation due to micro-organisms is the most frequent form that occurs in children. The *secondary* catarrhs occur with the lesions of more pronounced degree which belong to the causes. The catarrh, and hence the diarrhœa, that attends the ulceration of typhoid fever, the ulceration of dysentery, or that occurs in Bright's disease, and the diarrhœa that attends carcinoma or other organic disease of the bowel, is of this nature. In addition a catarrh of the bowels arises from venous stasis in the mucous membrane, with chronic congestion. This occurs in the course of organic heart disease or in disease of the liver with portal congestion.

Diarrhœa is a symptom of certain poisons, such as mercury, arsenic, and other corrosive agents. According to Brunton and others, the diarrhœa which occurs from the irritant action of food products and in cholera infantum is due to a toxic ptomaïne.

Diarrhœa sometimes fulfils a vicarious office. This is the case with the diarrhœa which comes on in cases of chronic Bright's disease and in acute Bright's disease before the supervention of uræmia. When diarrhœa occurs in a person with pallor, dimness of vision, and œdema, the urine should always be examined.

The Symptoms of Diarrhœa. Increased movements of the bowels. The frequency of the movements varies with the cause. In the diarrhœa of nervous origin, usually after five or six movements have occurred, the patient is relieved because the cause for the nervousness has disappeared. In catarrhal diarrhœa the number varies from half a dozen in twenty-four hours to the same number in an hour. Indeed, in some severe cases the evacuation may be almost constant.

Abnormal character of the movements. The movements may be (1) *fæcal*, with a small amount of *water*. They are light in color, softer than natural, but yet retain their form. They are the kind of movements seen in simple catarrh.

2. The fæcal matter is mixed with *undigested food*. The fæces are in scybalous masses, and the watery element is increased. They are the stools of the so-called dyspeptic diarrhœa.

3. Along with the fæces *mucus* in more or less degree is seen. The amount of mucus depends upon the seat of the inflammation as well as the intensity. Inflammations of the large intestine are attended with

mucous discharge. The mucus is not difficult to recognize. It may be mixed with and stained by fæces so that only by close inspection is it recognized. In milder degrees of catarrh it is seen on the surface of the fæcal masses.

4. The fæces disappear almost entirely, and instead the evacuations are *watery*. The watery evacuations may be discolored, as in the pea-soup evacuations of typhoid fever, or they may be almost clear water, as in the rice-water discharges of cholera.

5. The evacuations may contain *blood*. Bloody discharge usually accompanies mucus; when the catarrh is in the lower bowel it may occur independently of the mucus. If with the mucus, it tinges it in reddish specks, or small amounts of free blood are seen. The blood may be bright in color, and then usually comes from the rectum. The source of the blood may be, it must be remembered, from hemorrhoids, or fissure, which is unduly irritated by the diarrhœa. It is then bright red and unmixed with the movement, and from its position can readily be seen to have followed it. If mixed with the movement the blood may be black, as in all forms of *melæna*, or it may be dark red in color. The black blood usually comes from the small intestine, or stomach, and may be the result of ulceration in the stomach or even the swallowing of blood. On the other hand, it may be due to cirrhosis of the liver, with venous congestion. It may be due to the ulceration of typhoid fever and the intense inflammation of enteritis. It is a symptom of carcinoma of the bowel and is of frequent occurrence, almost pathognomonic in intussusception. It must be remembered that blood of this character is discharged from the bowel independently of diseases of that tube, as in purpura, scurvy and other blood diseases. (See Arteries of the Intestine, page 525.)

Microscopical and Bacteriological Examination. In simple catarrhal inflammation of the tubules, on microscopical examination but little is found except an excess of epithelium from the mucous lining. In more intense inflammations, in addition to epithelium there are pus and blood and mucus. Micro-organisms are found dependent upon the cause of the diarrhœa. In health Booker has found at least forty varieties of micro-organisms, many of which, in all probability, are not pathogenic. In health the bacillus coli communis and the bacterium lactis aëriformis are found. In the diarrhœa of children both forms are present in excessive numbers, because conditions favoring their growth arise, and in all probability are the cause of the irritation of the bowel. In that form of inflammation of the bowel known as dysentery, in addition to the bacteria that attend inflammations, the amœba coli is present. It has been found that dysentery may be due to a number of causes, but that the so-called tropical dysentery is due to the protozoa first described by Kartulis and in this country by Osler. (See Fæces.)

The symptoms that attend increased movement of the bowels depend upon the cause and also have direct relationship to the frequency of the evacuation. The symptoms most frequent are pain, flatulent distention, with borborygmi and tenesmus. *Pain.* The pain depends largely upon the cause. If the irritant is a product of indigestion, or a bulky mass, pain is more or less severe. It is situated in the centre of the

abdomen, and may extend all over. It occurs before the evacuation; it is sharp, lancinating, and is usually relieved by the movement. If the inflammation is in the large intestine the pain may be complained of in the course of the large bowel or be more intense over the cæcum and the sigmoid flexure. The rectum may be the seat of pain or of painful sensations. This has been described as a feeling of a hot ball in the lower pelvis. *Flatulent Distention.* The flatulent distention is not very great generally. The abdomen is distended, tympanitic on percussion, and tender on palpation, both of which may be more marked in the middle of the abdomen if enteritis alone is present, or it may extend along the course of the colon, as in the so-called entero-colitis of children. With the distention there are borborygmi. The rumbling usually subsides after the evacuation.

Tenesmus occurs in all forms of diarrhœa if the evacuations have been frequent. After the discharge of the contents of the bowel, particularly if from the rectum, the tenesmus is much more severe, and may be of constant occurrence. In the severe cases the tenesmus may be almost continual. On account of it prolapse of the bowel is liable to ensue.

General Symptoms. The *general symptoms* that attend diarrhœa depend upon the cause. In simple diarrhœa there may be slight feverishness only, with a little weakness. In diarrhœa, with excessive movements, with mucus, with or without blood, the fever is marked and may rise as high as 103°. The fever that attends dysentery is high, and usually rises rapidly at the beginning.

Prostration. More or less prostration attends all cases. It is, however, more marked when there are frequent watery evacuations. In its most pronounced degree it is seen in cholera and cholera infantum. *Collapse* rapidly ensues under these circumstances on account of the depleting effects of the excessive watery discharge. In catarrh of the intestines secondary to typhoid fever and other conditions the general symptoms depend upon the primary disease.

Chronic Diarrhœa. Chronic diarrhœa may be due to chronic inflammation of the bowels, as in chronic intestinal catarrh. It may be secondary to the ulceration of dysentery, tuberculosis, syphilis, or cancer. It is the common diarrhœa of amyloid disease. In chronic diarrhœa the stools vary, but seldom amount to more than ten to fifteen in a day. In chronic intestinal catarrh three or four movements occur in the twenty-four hours. They usually occur in the morning, the first evacuation taking place immediately on rising and the remainder during the morning hours. They are more common in women than in men, and are readily excited by exhaustion or nervous influence, as grief, emotion, or excitement of any kind. The stools are fæcal and watery and contain some mucus. The mucus usually coats the surface of the fæces. The color of the fæces is not changed. The patients usually suffer from the symptoms of intestinal dyspepsia or are subject to some gastric neurosis. They are not under weight, and except for the inconvenience of the morning hours could attend to the ordinary demands of life. They are more nervous than most people, and are liable to attacks of hemicrania.

Membranous Diarrhœa. In a number of cases the discharge from the bowels resembles membrane. The disease is also called membranous enteritis. The discharges contain much mucus, and may be a little more watery. After the fæces have been passed membrane is discharged. This may be in shreds or large masses, and may also be like a cast of the bowel. The patients are usually females who are hysterical and have some menstrual disorder. Pain may precede the discharge, and continue until there is complete relief.

Constipation. Constipation may be due to a number of causes. It may be due to alteration or diminution in the secretions of the intestinal tract, as is seen in all fevers, except when they are attended by specific intestinal catarrh, as in typhoid fever. Such diminution of secretion occurs in the summer, when there is more free perspiration than in other seasons, and is present in affections attended by excess of perspiration, or exhaustive diuresis. Constipation, therefore, is a common symptom of diabetes.

In addition to alteration of the secretion, diminution in the sensibility of the nerves may exist. This is the one chief cause of habitual constipation that is so prevalent. On account of carelessness the patient loses the habit of having a regular movement of the bowel each day, and in consequence the usual stimulus is removed. Constipation also occurs from weakness of the muscles.

The three conditions, diminution or alteration in the secretions, debility of the muscles, and impairment of the sensibility of the nervous mechanism, are combined influences on account of which constipation is so prevalent in persons of sedentary habits and in persons living upon improper diet. General diseases and local disorders which influence either of the above elements cause constipation. Thus in anæmia and chlorosis, in neurasthenia and hysteria, constipation is a common condition. Its occurrence in fevers has been mentioned. In the convalescence from exhausting disease and prolonged confinement to bed constipation is liable to ensue.

Local Causes. Atony of the abdominal muscles or of the bowel is the cause. Atony is most strikingly seen in peritonitis and typhlitis, in both of which a paretic state of the bowel develops. It is seen in the aged and in cachexia along with atony of other muscles. Obstruction of the bowels, acute or chronic, causes constipation usually (*q. v.*). If the obstruction is not complete there may be, on account of catarrhal inflammation, diarrhœa. Constipation often occurs on account of pain, seated in the rectum particularly. The pain is such that the patient shrinks from an evacuation. Frequent postponement soon causes constipation. The pain may be due to fissures, to hemorrhoids, or to fistula. Constipation occurs also from local diseases in other portions of the body influencing, in all probability, the nervous mechanism by which peristaltic action is excited. In acute and chronic disease of the brain and cord, as meningitis and myelitis, constipation is a chronic attendant. It also occurs in tetanus. If the bowel is deprived of fæcal matter evacuations of the bowels cease. Constipation is a constant sign of stricture of the pylorus and of stricture or cancer of the œsophagus.

Symptoms of Constipation. Constipation is characterized by diminu-

tion in the frequency of the bowel movements. The frequency of the movements varies in health. Some persons are comfortable with an evacuation taking place once a week, or at least every third or fourth day. There are cases on record in which the evacuations took place but once a month. Cases of this class are usually due to muscular paralysis of the bowel, with secondary dilatation. The accumulation of fæces is removed by a sharp attack of diarrhœa, attended by much pain. The diarrhœa sometimes continues for twenty-four hours. When it sets in fever may be present until there is thorough evacuation.

Local Symptoms. Usually the symptoms that attend constipation are local on account of the discomfort of the accumulation of fæces. The local symptoms may be limited to the rectum or extend through the abdomen. In the rectum there is a sensation of a mass, which may cause some pain. The abdomen is distended; there is considerable rumbling, and sometimes peristaltic waves are seen. The accumulation of the fæcal mass in the bowel may set up tormina and tenesmus, and portions of the masses may be discharged from time to time. In other words, a diarrhœa may occur, the diarrhœa of constipation, or spurious diarrhœa. The stools are small, composed of hard scybalous masses, generally coated with mucus, and with some blood. The evacuation does not give relief, and the desire for a movement may be more or less continuous.

On examination in constipation with fæcal accumulations the outline of the colon may be marked out by palpation and percussion of the distended abdomen. In its course masses are felt varying in size from a marble to a base ball, and in consistence they may be soft to the palpating finger; they are never indurated like a calcareous mass, as gall-stones or a mass due to malignant disease.

General Symptoms. While in many instances the general symptoms are of no consequence, in others the patients are nervous and may be in more or less impaired health on account of the secondary effects upon the stomach. Digestion is impaired and the form of indigestion is that which attends neurasthenia.

The patients are of spare habit, usually of dark or muddy complexion. They may be depressed. There is inaptitude for mental exertion; they are more or less hypochondriacal. The tongue is constantly furred, the appetite variable; there is weight and fulness after eating, and generally some flatulency.

The Secondary Effects of Constipation. The effects of constipation upon the intestines is various and sometimes disastrous. They are dilatation and ulceration. The former may become enormous, as in cases reported by Formad and Osler. The dilatation may be so great as to distend the entire abdomen. The ulceration may be localized to the rectum, or cæcum, or extend throughout the entire large intestine. On palpation the course of the colon is tender, and fæcal masses may be outlined and may be painful because of their pressure upon the adjacent ulcer. In the rectum the ulcer may be deep, and be followed by peri-rectal abscess.

In the cæcum the accumulation may be such as to cause a large boggy swelling, extending in the course of the cæcum, which is tender on pressure, and dull on percussion. Stercoral typhlitis is caused (*q. v.*).

Fæcal impaction, with secondary ulceration, is of frequent occurrence in typhoid fever. This must be borne in mind, for often serious general and local symptoms arise because it is overlooked. Recently I saw a case with the diarrhœa of constipation, with some fever, which persisted for weeks after the usual course of typhoid fever. It was thought the patient had tuberculosis, or that the typhoid process was abnormally prolonged. Examination disclosed ulceration into the vagina, and the fæces were constantly discharged from this orifice. It had been thought that the discharges of fæces were due to diarrhœa. Of course, fever attended the process, and rendered the case all the more obscure.

In this connection must be mentioned the constipation that occurs on account of lead-poisoning, and the exhibition of drugs, as opium, or astringents. The constipation of lead-poisoning is usually attended by colic, and the blue line on the gums is seen, while wrist-drop or other manifestation of lead may be present.

Intestinal Hemorrhage.

The causes are general and local. The general causes are those that accompany hemorrhage in other localities. (See Gastric Hemorrhage.) The local causes (1) in which hemorrhage is small, are: inflammation of the bowel; traumatic injury to the bowel from hernia, fæces and parasites, and foreign bodies swallowed, or from corrosive poison; tumors of the bowel; as in cancer, invagination, and ulcers. (2) Large hemorrhage occurs in the congestion attending portal obstruction and liver disease, or disease of the heart with secondary obstruction. Aneurism of the superior mesenteric artery, or aneurism rupturing into the intestine, and occasionally embolism of the artery will be followed by intestinal hemorrhage. It occurs in ulcers first from typhoid fever; second, from dysentery; third, from syphilis. It may occur in pyæmia and septicæmia, or the acute exanthematous diseases. The symptoms may be those of hemorrhage alone: collapse, pallor, failure of sight, tinnitus, vertigo, small pulse, and general restlessness. The hemorrhage must be copious under these circumstances, and is due (1) to the bleeding of an ulcer, as in typhoid fever; (2) to portal obstruction; (3) to an aneurism; (4) to purpura or hæmophilia.

A second group of symptoms referred to the appearance of the discharges from the bowels. The stools are bloody; if the hemorrhage is low down they are bright red and usually mixed with fæces. If high up, they are tarry. They are known as melæna (see Fæces).

The passage of the stools is preceded by colicky pains, or there may be some rumbling. The diagnosis must be directed toward determining the cause of the hemorrhage, as well as its seat; the history, the associate diseases, or symptoms, aid in determining the cause. Examination of the rectum may afford a clue to its origin.

The Data Obtained by Observation. The Objective Symptoms.

Physical Signs. *Inspection.* Local and general enlargements of the abdomen have been discussed in the preceding pages. Movements

of the intestines are seen in obstruction due to increased peristalsis. The intestine above the point of obstruction may swell into a defined tumor which becomes hard and dull, tympanitic on percussion.

Palpation. Tenderness, peristalsis, peritoneal friction, the bubbling of gas through a constriction of the bowel, and tumors, are recognized by palpation. It is necessary often to place the patient on all fours or in a knee-chest position.

Percussion. The normal note is tympanitic. Local areas of dulness may be due to intestinal tumor. Light percussion should be employed. A dull tympany indicates a solid mass surrounded by the distended intestines. The outline of the large intestine can be ascertained by filling it with water.

The Fæces. General Considerations and Macroscopical Appearances. The number of stools varies chiefly in health with the individual and the character of the food taken. After infancy, one passage in twenty-four hours is the rule, but it is natural for some persons to have two or three, and for others to have but one passage in two, three, or four days. Such a condition is termed constipation, while pathological constipation is properly called obstipation. The opposite condition is known as diarrhœa. The amount and character of food and drink ingested influences the number of the stools. Exercise also plays a rôle; and increased or diminished peristalsis, from whatever cause, will induce on the one hand diarrhœa, and on the other constipation. In disease the greatest extremes are met with—from the non-passage of fæces for days, as in obstruction, to an almost continuous discharge, as in some forms of intestinal inflammation. It is well to remember that diarrhœa may be the symptom of obstipation, as when impacted fæces in typhoid cause looseness of the bowels.

The *amount* of fæces varies with the quantity and nature of food. If most of the food is digested and carried away for the economy, there will be but little left to form fæces. In any disease that prevents the absorption of digested food, or causes an increase in the fluid contents of the intestine, as cholera, the amount of fæces will be increased. In health about 140 to 200 grammes are voided in twenty-four hours.

The *form* and *consistence* of healthy stools varies somewhat. They are commonly cylindrical and firm or mushy. When they remain long in the intestinal canal, and the water is extracted, they become hard and may form balls, or flattened masses, known as scybala. These are frequently seen in convalescing typhoid patients. On the other hand, the fæces may be without form, and are then liquid, either watery as in cholera, or purulent or bloody. Many varying diseases cause such a condition.

The *odor* of fæces is sometimes more or less characteristic of certain conditions. Thus the stools of nursing infants have a sour smell, while in infantile diarrhœa and when fermentation takes place they have an odor of sebacic acid. When urine is mixed with the passage the odor will be ammoniacal; with blood present it often has a stale odor.

The *reaction* is not constant. Thus in intestinal catarrh, with acid

fermentation it will be acid, or in alkaline fermentation it will be alkaline.

The *color* of the stools varies too much to be of special diagnostic value. In health it is light to dark brown, due chiefly to the presence of hydro-bilirubin, a product of decomposition of bile pigment which is never normally found unaltered in the fæces. It is influenced greatly by food and medicines. When certain berries, as huckleberries, are eaten, or certain medicines taken—iron and bismuth—they make the passages black. Calomel causes green stools, by causing biliverdin to be present. Green stools may also receive their color from the presence of a bacillus which produces a green dye. Santonin, rhubarb, and senna, cause yellow; and hæmatoxylon, red stools. The last fact is important, as parents or nurse should always be instructed, when hæmatoxylon is given, to expect red passages.

The fæces may be red or reddish from the presence of unaltered blood, or black when the blood has undergone changes; the so-called "tarry stools" are of this character. With a decrease in the amount of bile the stools become less colored, and if the bile is cut off they become clayey. This color may in cases be due to the presence of fat left undigested because of the lack of bile. On the other hand, if from disorders of the stomach and intestine the intestinal contents pass too rapidly through the intestine, the fæces may contain unaltered bile or unchanged bile pigment, giving a green or yellow color, and showing the bile reaction.

The *constituents of fæces* that can be recognized by the naked eye are numerous. Seeds, stones, and skins of fruit and berries; fibres of vegetables, are often seen in healthy stools. In the passages of children and weak-minded individuals may be present foreign substances of all descriptions. Foreign bodies and partially digested portions of food may be mistaken for parasites. Portions of tumors from the digestive tract may appear in the fæces.

In certain diseases of the stomach and small intestines, and in those who eat very fast and do not properly masticate their food, undigested and unchanged particles of food may be seen in the stools.

Shreds of mucous membrane of varying size are passed with the fæces, or constitute them, in cases of membranous enteritis. Von Jaksch saw such a shred 5 cm. long and 3 cm. broad in a case of cholelithiasis.

Particles resembling sago-grains, perhaps the result of over-indulgence in farinaceous food, have been met with.

Gall-stones in the fæces have great clinical value. They may escape detection, if not properly sought for. When suspected, each passage should be passed through a linen sieve, the fæcal masses being softened with water. They may be found as small, crumbling masses, composed chiefly of cholesterin (intra-hepatic calculi), or as hard, irregular, smoothly-worn, shining, many-sided, hard stones, sometimes as large as an egg; usually the size of a pea. Enteroliths are occasionally seen. They are said to originate in the appendix.

Blood may be present in the fæces in varying proportions and conditions. When found unaltered on the surface of scybalous masses it is from the rectum or large intestines, and probably the result of trauma-

matism. Hemorrhoids, if bleeding, may cause such an appearance, or may cause very free hemorrhage. Severe hemorrhage may come from ulceration of the rectum or colon, due to malignant disease or severe inflammation. The blood may be intimately mixed with the fæces, and have its origin in the large intestine, but much more commonly it indicates a source in the stomach or small intestine. Under such circumstances it is nearly always more or less changed by the intestinal juices, and is brownish-red or black, the tarry stool as mentioned above, or has the appearance of coffee-grounds. The more retarded the passage the greater the change; while, if quickly expelled, blood from the small intestine may be passed unchanged, as in the hemorrhage of typhoid fever. The microscope detects blood when the naked eye fails to detect it. It is to be remembered that certain drugs, as already stated, may color the fæces red, and simulate blood.

Mucus may be present in the passages in health, but when in any marked quantity there is a catarrh of the mucous membrane of the intestines. When hard scybala are covered with mucus, or the mucus is seen as shreds, the large intestine is the seat of a catarrh, though it may be mixed with thin stools, as in dysentery. But usually when the mucus is finely divided and mixed with the fæces, it comes from the small intestine. Mucus shreds have already been mentioned. In cholera the particles of mucus look like boiled rice, hence the term "rice-water stool."

Fatty stools, to the naked eye, appear greasy or even clayey, when there is much fat, even though bile pigment may be present.

Pus may be present in large quantities from rupture of an abscess into the intestinal tract, or when there are ulcerations from various conditions, producing pus in considerable quantities.

Many animal parasites are visible to the naked eye, but a full consideration of them will be given in the following paragraphs.

Microscopical Examination of the Fæces. A small portion of the solid fæces to be examined is placed on a slide moistened with water and $\frac{1}{2}$ per cent. salt solution, and a cover-slip applied; or if liquid, various drops are to be examined. The different constituents to be found will vary with the food taken as well as with disease.

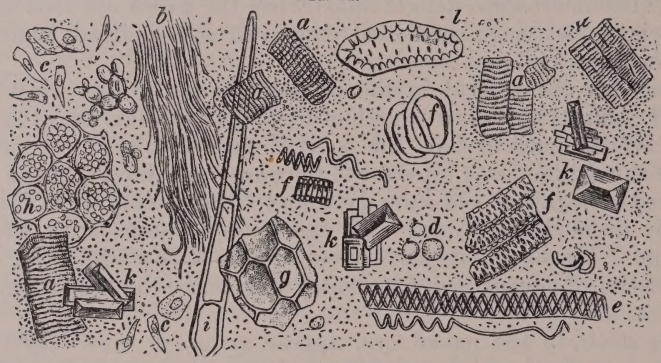
Constituents Derived from Food. There may be portions of digested or undigested food. In general it may be said that the presence of large pieces of unchanged food or many small particles of undigested or only partially digested food, indicates defective digestion in the stomach or small intestine. If unchanged bile is present, then particles will be colored yellow, another indication of diseased functions.

From the *food* we may see muscle and elastic fibres, more or less as the quantity of meat the patient eats varies. The former are recognized by their transverse striation; the latter, by their double contour and curling ends. Fat may be present as fatty globules or in the form of needles, fatty crystals. Much fatty food increases their number, and they are seen plentifully in alcoholic poisoning, in jaundice, in the fatty pancreatic diseases, tuberculosis of intestines, diseases of the mesenteric glands, and enteritis. The crystals may be transformed into fat-drops by the addition of acid and heat. When meat is eaten freely, areolar tissue

may be present, but its presence otherwise points to defective digestion. Various forms of vegetable cells are commonly seen, in which granules of starch may be contained, or the starch particles may be free. Undigested milk occurs in the stools of children and when diarrhœa prevails; a substance, probably cercin, has been described by Nothnagel as occurring in fæces of persons who have intestinal disturbances.

In persons living on vegetables, the majority of the above constituents will be absent, and in infants who partake only of milk, the derivates of meat are absent, while there will be an excess of fatty crystals and fat globules and coagulated products.

FIG. 81.



Collective view of the fæces. (Eye-piece III., objective 8A, Reichert) *a*. Muscle fibres. *b*. Connective tissue. *c*. Epithelium. *d*. White blood-corpuscles. *e*. Spiral cells. *f-i*. Various vegetable cells. *k*. Triple phosphate crystals in a mass of various micro-organisms. *l*. Diatoms. (VON JAKSCH.)

CONSTITUENTS FROM THE ALIMENTARY TRACT. *Epithelium.* In every normal stool will be found epithelium of the squamous variety. Occasionally the columnar form is seen, and altered epithelial cells are very common. In intestinal catarrh their number is greatly increased.

Red Blood-corpuscles. In the majority of blood-stained stools red blood-cells are not found; in their stead will be seen masses of free blood-coloring matter and rhombic crystals of hæmatoidin. Red cells are seen in dysenteries, in bloody stools in which the blood comes from near the anus, as in hemorrhoids, and when a hemorrhage is discharged with the fæces soon after its occurrence. If there is any doubt as to the presence of blood when the corpuscle cannot be found, a true decision can be reached by examining for hæmin crystals, according to Teichmann's method. A portion of fæces is dried and powdered, placed on a slide with a grain of common salt, and covered by a cover-slip. A few drops of glacial acetic acid are directed beneath the slip, the slide is heated just to boiling, and if blood has been present, reddish-brown rhombic crystals of hæmin will soon be found.

Leucocytes. These bodies are frequently seen in healthy stools. When pus is found in or discharged into the intestinal canal, they are found in great numbers, as in ulceration of the intestine and in abscess.

Molecular débris, or detritus, occurs in all fæces as part of the waste products.

Crystals. *Fat* crystals are the most important. They have been quite fully considered above. There seems to be but little doubt that the crystalline needles found in the fæces are salts of fatty acids, and not tyrosin.

Charcot-Leyden crystals, similar to those already described under sputum, have occasionally been met with in the stools of typhoid fever patients, in dysentery, intestinal tuberculosis, and ankylostomiasis.

Hæmatoidin crystals occur as reddish-brown, hard, needle-shaped bodies, usually in clusters, and free or enclosed in masses of mucin or a substance resembling it. They have been found in the fæces of breast-fed infants; in cases of chronic intestinal catarrh, and by Von Jaksch in the stools of a case of nephritis.

Crystals of various *salts of calcium*, of *triple phosphate* and *cholesterin* will often be recognized, but they have no diagnostic value. When bismuth is being administered black rhombic crystals of the sulphide of bismuth will be recognized.

PARASITES. (*A*) *Animal*, and (*B*) *vegetable* parasites flourish in the intestinal tract, and the presence of some of these in the fæces is of the greatest clinical importance.

A. ANIMAL PARASITES. Following Leuckart's classification, we will consider these parasites under the secondary heads:

I. PROTOZOA. 1. *Rhizopoda.* This variety is made important because the amœba dysenteriæ or amœba coli belongs to it.

(*a*) *Amœba Dysenteriæ.* *Amœba Coli.* This protozoön has been found so many times by various observers in different parts of the world that it can now be considered to be the causative factor of so-called tropical dysentery. The subject has received special study in our own country by Osler,[1] Stengel,[2] Dock,[3] and Councilman and Lafleur.[4] The unexcelled work of Councilman and Lafleur is at the present time the best that has been published in any country; and to it the reader is particularly referred. The following notes are based on this book.

The amœbæ dysenteriæ vary in size from 0.012–0.035 mm. They are found most plentifully in the small gelatinous masses often to be seen in the fæces. They vary in number in different cases and in the same case at different times. The severer the lesions the more numerous are the amœbæ. When not active they are round or oblong, and highly refractive. They contain one or more vacuoles of varying size. Occasionally the division into an ecto- and endosarc is easily made out. When thus inactive, they may be confused with swollen connective-tissue cells and compound granular bodies found in fæces. The active amœbæ have, however, a characteristic movement. This consists of progression and of thrusting-out and retraction of pseudopodia. Their activity varies greatly. It is best seen when the body heat is maintained. The stools should be passed into a clean and warm pan, and examined immediately, or kept warm until examined, and a warm stage should be used with the microscope. The division into ecto- and endosarc is

[1] Johns-Hopkins Hospital Bulletin, May, 1890, vol. i., No. 5.
[2] Phila. Med. News, 1890.
[3] Texas Med. Journal, April, 1891.
[4] Johns-Hopkins Hospital Reports, vol. ii., Nos. 7, 8, 9.

usually clear during activity. The ectosarc is composed of a hyaline homogeneous mass, as are the pseudopodia, while the endosarc is made up, not of granular matter, but of a dense homogeneous matter enclosing vacuoles and a nucleus. The vacuoles may vary in size as well as in number. There may be one or two large ones, or the entire endosarc may appear as made up entirely of small vacuoles. The nucleus is sometimes plainly seen as a small rounded body, but is more often difficult to distinguish from the vacuoles. Dried cover-slip preparations may be stained with the various aniline dyes, but the results are not satisfactory.

The amœbæ will often be found to enclose bodies such as red blood-corpuscles, pus cells, blood-coloring matter, bacilli and micrococci.

In examining the fæces for amœbæ dysenteriæ the suggestion given above concerning the warm bed-pan and warm stage to the microscope,

FIG. 82.

Amœbæ coli. (HALLOPEAU.)

and above all, the immediate examination of the stool, should be adhered to. The small gelatinous masses should be selected when present. Various magnifying powers should be used, including the $\frac{1}{12}$-immersion lens.

(*b*) *Monadines*, pear-shaped, with a long slender process, are seen alive only in perfectly fresh stools. They are not found constantly in any one disease.

2. *Sporozoa.* Under this head belongs the coccidium perforans of Leuckart. They are short, elliptical bodies, which infest the intestinal mucous membrane, and may damage it badly; they are often discharged in large numbers.

3. *Infusoria.* (*a*) *Cercomonas intestinalis.* This is a pear-shaped body, nucleated, with eight tentacles of varying length. It is found in the fæces of persons suffering from various diseases, as cholera and typhoid fever, and probably of itself causes diarrhœa.

(*b*) *Trichomonas intestinalis.* Larger than the cercomonas, and covered with ciliæ at the club end. It is not diagnostic, and is not common.

(*c*) *Paramœcium coli.* Larger than the preceding, 1 mm. long—oval, covered everywhere with ciliæ; may be found in diarrhœic stools.

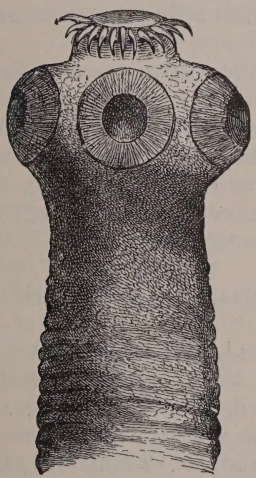
II. Vermes. These are much more generally known and are of much more clinical value than the preceding.

They have important clinical value, as the presence of some of them in the intestinal canal gives rise to many untoward symptoms. They will be considered under (*A*) *Platodes.* (*B*) *Annelides.*

A. Platodes. 1. *Tape-worm—Cestodes.* These parasites infest the small intestine only, to the walls of which they cling by the head. The head and neck are small; the joints are flat and form long ribbons. The distal joints continually drop off and can easily be recognized in the stools by the naked eye, and the eggs by the use of the microscope. The fæces are best washed in water and broken up to obtain the eggs. As the lower joints are lost new ones take their place from above. The more important are as follows:

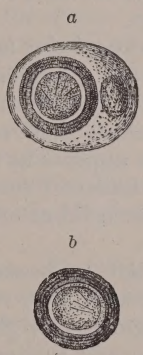
a. Tænia solium (Fig. 83.) reaches a length of two to three metres. The head is the size of a pin-head. The neck is 2.5 cm. long, as thick as a thread, and without joints. The segments forming the body are

Fig. 83.



Head of T. solium. × 45. (Leuckart.)

Fig. 84.



Ova of T. solium. *a* with yolk, *b* without yolk, as in mature segments. The hard brown shell is indicated. (Leuckart.)

short and broad near the neck, but as they increase in size there is more growth in length than in width. The average dimensions are 9 to 10 mm. × 6 or 7 mm. The head appears dark, the body white. The joints are easily detected in the fæces by the naked eye. Under the microscope the head is seen to be spheroid, with four pigmented sucking discs surrounding at the base a rostellum, which is a "crown of hooks"—chitin hooks—about twenty-four in number. In the ripe segments, or proglottides, is seen the longitudinal uterus with about twelve horizontal ramifications to a segment. The eggs are round or oval, 0.035 mm. long, with a thick, striated shell when ripe, and contain hooklets.

b. Tænia mediocanellata, or *saginata.* This worm is four or five metres long. The head is slightly larger than that of the T. solium,

and more pigmented, and the segments are longer, fatter, and darker. The head is supplied with four powerful sucking cups, but there is no rostellum or hooklets. The uterus in the ripe segments is much more finely branched than in the solium, and these segments have independent movement. The eggs are very similar to those of the T. solium, but may be rather larger.

c. Tænia nana. In length the T. nana is only 10 to 15 mm., and 0.5 mm. in breadth. The round head is but 0.3 mm. in diameter. The segments are all short, and at the lower end of the body are four times as wide as they are long. The head is found to have four round suckers at the base of a rostellum that can be inverted. At the base of the rostellum are about twenty-two hooklets. The uterus is oblong and filled with eggs. The eggs have a double membrane.

d. Tænia cucumerina. This parasite is found to be 5 to 20 cm. long and about 2 mm. wide. The head is placed at the thinner end, and under the microscope are to be seen some sixty hooklets distributed with order about the rostellum, and four sucking cups. The lower segments are decidedly larger than the upper—6 by 7 mm. When ripe, they become reddish, and contain cocoon-like bodies, in which are six to twelve eggs.

e. Bothriocephalus latus. This is the largest of the worms, measuring 7 or 8 metres. The head is somewhat drawn out, and on either side is a long, narrow sucker. There are neither hooks nor rostellum. The proglottides are short near the head, but become square further down. The uterus appears as a rosette, peculiar to this worm. The eggs are oval and measure 7 mm. by 0.045 mm., have a shell covering, with an opening like a lid at one end. Ripe segments are thrown off in bunches, not singly.

It will not be necessary to describe certain other varieties that are rarely met with.

2. *Trematodes,* or *flukes. a. Distoma hepaticum* measures 28 mm. by 10 mm., and is shaped like a leaf. A short head is situated at the broad end and has one sucker; on the under surface is another sucker, and between the two is the opening of the uterus, a highly convoluted arrangement. The eggs are brown, oval, about 0.12 mm. long, and have a lid at one end. It is not often seen.

b. Distoma lanceolatum. This round-shaped worm is about 8 mm. long and 3 mm. broad, and in other respects resembles the preceding. The eggs are more rounded and contain minute embryos. Like the D. hepaticum, it is rarely seen.

c. Distoma crassum is the largest—4 to 8 cm. long. These flukes are endemic in parts of Japan. In general these animals occupy the bile-passages or upper part of the small intestine.

B. ANNELIDES. 1. *Round worms—nematodes.* A. *Ascarides.*

a. Ascaris lumbricoides. This is the parasite usually referred to by the term round worm. It resembles the common earth-worm in shape and color. The male worm is about 250 mm. long and the female 400 mm. The head is made up of three prominent lips, and is supplied with microscopical teeth. The vulva of the female is in the posterior third of the body. The eggs are rounded, brownish, 0.06 mm. in diameter,

and covered when fresh by a rough albuminous coat over a hard shell. This worm has the small intestine for its habitat. It may pass with the stools or work its way into the stomach and be vomited (the writer has had them thus vomited during the etherization of a child of ten years). They have been the cause of jaundice by crawling into the ductus choledochus, and may infest the larger hepatic ducts. Enormous numbers may be present in the intestine at one time.

b. Oxyuris vermicularis. The thread- or seat-worm inhabits the large intestines, and is often present in the stool as a white, thread-like body; the male 5 mm. and the female 10 mm. long. They often wander out of the anus and into the vagina. The head has a number of small lips, and is covered with a thick skin. The female has one vagina and two uteri. The eggs are unsymmetrical, have a laminated shell, and have a diameter of about 4 mm.

B. *Strongylides. Ankylostomum duodenale.* This is a round worm, reaching a length of 6 to 10 mm. in the male and 10 to 18 mm. in the female, and can therefore be seen easily, though the eggs are much more frequently found in the stool than is the worm itself. With the eggs there may be present in the stools large numbers of Charcot-Leyden crystals. The head is prominent, especially in the male. Four hook-like teeth surround the mouth, and by these the animal attaches itself to the intestinal wall. The tail of the male is expanded and that of the female pointed. The vulva is in the posterior third. The eggs are oval, about 0.05 mm. in diameter, and contain one to four cells—embryonic globules, which rapidly develop in a warm place outside the body, and may thus be recognized. The worm infests the small intestine, especially the jejunum. It often causes serious symptoms—bloody stools and intense anæmia.

C. *Trichotrachelides. a. Trichocephalus dispar.* The whip-worm is 4 to 5 cm. in length, the female being longer than the male. It is recognized by the contrasting form of the anterior and posterior portions. The former is thin and threadbare, the latter expanded and broad, and in the male curled up. The eggs are brownish, about 0.05 mm. long and half as broad, and have a button-like projection at either end; they are to be recognized in the stools, where large ones may be present. There may be only a few, or thousands, of the forms present in the body. They live chiefly in the cæcum and large intestine. They have been thought to cause beri-beri by some writers.

b. Trichina spiralis. It is the adult trichinæ which exist in the intestine, and are found very infrequently in the fæces. These produce the embryos, which become muscle trichinæ. The adult male is 1.5 mm. long and the female twice that length. The former has two projections from the hinder end, between which are four papillæ. The female has a tubular uterus and a tubular ovary in the posterior half of the body.

D. *Rhabdonema. Strongyloides.* Under rhabdonema intestinale we now include two small nematodes, which were termed anguillula intestinalis and A. stercoralis, and which are probably one and the same. They are found in the stools of cases of endemic diarrhœa of hot countries. Usually the young embryos, which have developed in the intestinal canal, are rejected with the stools. These sexually mature

embryos are 0.8 to 1.2 mm. long, male and female respectively. They are round and have a cone-shaped head. There are two jaws and two teeth in each. The adult worm is about 2.2 mm. long and 0.04 mm. thick. The mouth has three lips. The vulva is at the beginning of the posterior third. The eggs might be easily confounded with those of the ankylostomum duodenale but are somewhat more pointed—larger. The rhabdonema infests the small intestine, and is frequently found in connection with ankylostoma.

Ecchinococcus hooklets and portions of the striated cyst wall have been found in the fæces. The rupture of an hydatid cyst into the intestine may have much clinical value when the above structures are found—pointing to a cyst in the abdominal cavity.

B. Vegetable Parasites. We find both (I.) *pathogenic* and (II.) *non-pathogenic* vegetable parasites in the fæces. The latter we have classed as (1) moulds, (2) yeasts, and (3) fission fungi.

1. *Moulds.* The only mould found in the stools is the thrush fungus, when children are the subjects of thrush in the mouth. It is of very rare occurrence in the fæces and has no special clinical import.

2. *Yeasts.* In all fæces, in health or disease, yeast fungi exist. They are most numerous in acid stools. They are round or ovoid and usually occur in groups. They stain dark brown with a solution of iodine and iodide of potash, while apparently similar cells become violet or blue with the same dye.

3. *Fission Fungi.* Bacteria are found in greatest numbers in the fæces, chiefly as bacilli, micrococci and spirilla. They may be grouped as torulæ or sarcinæ. They present active movement and may be separate or in colonies. The bacillus coli communis (*B. termo*) is the most frequent form met with, both in health and disease. It is not yet determined what relations it holds to normal and abnormal conditions, or what is the true relationship between it and certain other bacteria. B. subtilis is another bacterium found both in health and disease. As above stated, there are various organisms which stain brown with iodo-potassic-iodide solution, and others which become blue with the same dye. Von Jaksch has studied these latter closely. They take various forms, as long or short rods, and take different shades of blue or violet. One of them is the clostridium butyricum of Nothnagel. It occurs as large round cells, like yeast fungi, and stains like the tubercle bacilli with the Ziehl-Neelsen fluid. Von Jaksch finds these fungi in greater abundance in intestinal catarrh. They are present in both acid and alkaline stools.

Bacillus Coli Communis has been found in the blood, various organs, fæces of cholera patients, in healthy fæces, in the air, and in putrefying infusions; it also can be found in the peritoneal exudate in most cases of peritonitis.

Morphology. A bacillus, 4 to 6 μ by 2 to 3 μ, with rounded ends, sometimes in cultures a short oval. Five or more filaments have been observed.

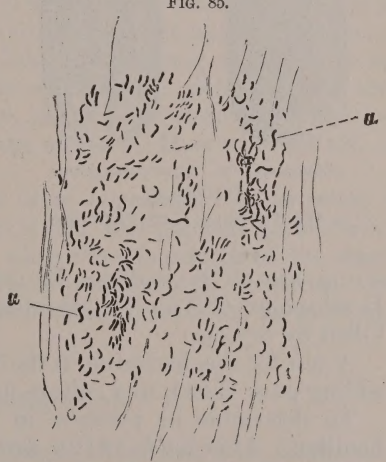
Biological Properties. Aërobic; facultative anaërobic; non-liquefying; as a rule, non-motile.

Growth. On gelatin plates the colonies vary very much. The deep

colonies are transparent straw color to dark brown, or may be granular and opaque. The surface colonies are large and spherical, centre dark brown, edges transparent. In stab cultures the surface growth is thin and dry. There is abundant growth along punctures, which is white by reflected but amber by transmitted light; sometimes moss-like tufts are seen. On potato, a soft shining brownish yellow layer grows. Stains with anilines, but not by Gram's method. Injected in guinea-pigs, it produces fever, diarrhœa and collapse. Injected into abdomen of rabbits, causes a typical peritonitis.

Pathogenic Fungi. SPIRILLUM CHOLERÆ ASIATICÆ. The *Comma Bacillus.* The comma bacillus of Koch is the specific causative agent of cholera. In a disease so widespread in time of epidemics, and so fatal, it is of great importance to be able to recognize the bacterium that produces it. Works on bacteriology give a fuller study than is permitted here, and should be consulted. This is more especially true because, while the bacilli, as found in the stools, can be stained quite easily, and may be recognized by expert microscopists, in the great majority of cases their recognition is only effected by bacteriological examination. They have no specific relation toward dyes, as have tubercle bacilli. The cholera bacillus is a short, more or less bent rod, both shorter and thicker than the tubercle bacillus, and generally shaped like a comma. They are often found placed end to end and thus form a curve like a spiral. They are always present in the stools of cholera patients and sometimes in the vomit. They are particularly abundant in the mucous floccules of the rice-water discharges, and can be obtained from the linen soiled by the same. Cover-slip preparations are made from these portions by placing a uniform film on the slip, drying it in the air, and then passing it through the flame of a bunsen burner or spirit lamp.

FIG. 85.



Cholera spirilla grown on moist linen. × 600. (After KOCH.) Cultivated from the dejections after two days.

The spirillum or so-called "comma bacillus" consists of a slightly curved rod, with rounded ends, 0.8 to 2 μ long by 0.3 to 0.4 μ broad. It is usually slightly curved like a comma, but may form a half-circle, or two may be joined like an S. Under certain circumstances they grow out into long spiral threads. By Löffler's method a single flagellum is found on the rods. It stains with anilines, but slowly. An aqueous solution of fuchsin is best. (See Plate I., Fig. 3, A; and Fig. 85.)

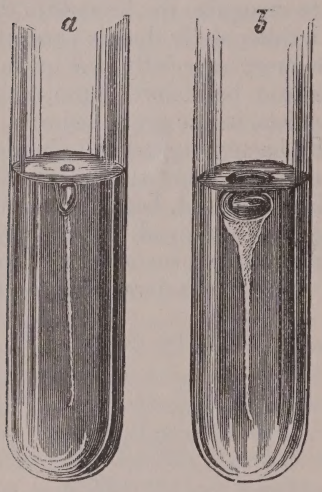
Biological Properties. Aërobic (fac. anaërobic), motile, liquefying.

Growth. Grows in ordinary media at room temperature; faster in oven. Does not grow except between 14° to 42° C. *Gelatin plates:* At the end of twenty-four hours small white colonies appear deep in the

gelatin. These grow toward the surface and liquefy the gelatin in a funnel form, which gradually deepens, and at the bottom the colony is seen as a small white mass. Under low power the colony is white or pale yellow, margins uneven, texture granular, surface looks as if covered with bits of glass. When liquefaction begins a dim halo forms about the colony, which by transmitted light is roseate in hue.

Stab Cultures in Nutrient Gelatin. Develops all along the puncture, liquefaction beginning near the surface, forming a funnel which enlarges, and finally the gelatin almost entirely liquefies. (See Fig. 86.) On potato, a thin transparent grayish-brown layer. Milk, bouillon, blood-

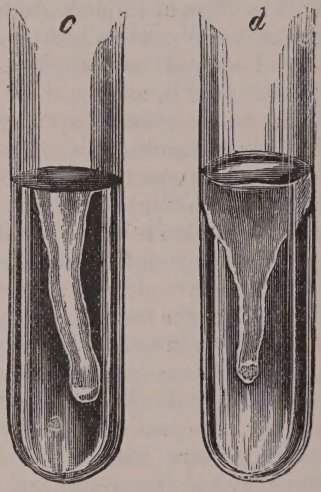
FIG. 86.



Cholera spirilla. Tube cultivations. (FLÜGGE.)

a, after two days; *b*, after four days.

FIG. 87.



Finkler and Prior's comma bacillus. Cultivations in gelatin.

c, two days; *d*, four days old.

serum, are all favorable. In media with other bacteria it soon dies. Death-point, 52° 5′. In moisture it retains vitality for months, but is killed by drying.

A test for this bacterium is the development of a purplish-red color on adding pure H_2SO_4 to a culture in nutrient gelatin.

To determine its presence in the shortest time, inoculate diluted bouillon. After ten to twelve hours a wrinkled film has formed. Make another culture in the same way from this, then inoculate gelatin plates, and use color test on these. Several toxins have been isolated.

The bacillus of cholera nostras and one found in cheese by Deneke resemble the comma bacillus in shape, though somewhat larger, but they have bacteriological peculiarities by which they can be differentiated.

SPIRILLUM CHOLERA NOSTRAS. *Morphology.* Longer and thicker; central part thicker than ends. Stains same as above.

Biological Properties. Culture. A thick, stocking-like funnel of liquefaction instead of a fine, straight funnel. (See Fig. 87.)

Typhoid Fever Bacillus. This bacillus is present in the stools of typhoid-fever patients, but cannot be directly differentiated by microscopic examination alone, either when stained or unstained. It is necessary, for its detection, to make pure cultures according to bacteriological methods. The bacillus is about as long as the tubercle bacillus but much thicker, being one-third as thick as it is long. The ends are rounded. It is best stained by concentrated aqueous solutions of methylene-blue, the dried preparations on the cover-slip being prepared as above. (See Plate I., Fig. 6, B; and Typhoid Fever.)

Tubercle Bacillus. The bacillus of tuberculosis is frequently found in the fæces of persons suffering from intestinal tuberculosis and occasionally in the fæces of cases of pulmonary tuberculosis, when sputum containing bacilli has been swallowed. When tubercle bacilli are constantly found in the fæces, and in large quantities, it points to the former condition almost to a certainty. They are detected in the same manner as tubercle bacilli in the sputum.

Bacilli of Booker. No less than nine bacilli have been described by Booker. Each of these has been found by him in cases of diarrhœa in children. Seven of these resemble very closely bacillus coli communis. Bacillus A is a bacillus with rounded ends, 3–4 μ by 0.7 μ. It is aërobic and facultative anaërobic, liquefying, and motile. Colonies on agar and potato are dirty brown. On gelatin they liquefy too soon to show characteristic form.

This bacillus is found in the stools of cholera infantum.

Chemical Examination. The *chemical examination* of the fæces is of but slight clinical value. Mucin and albumin are normally present; peptones, in different diseases (Von Jaksch). Among the acids to be found are bile acids, volatile and fatty acids, formic, acetic, butyric, and propionic acids; while phenol, indol, skatol, cholesterin, and fats are always present, according to the same author. They will not aid in diagnosis.

The normal coloring matter of the fæces is urobilin; its presence is shown by the proper tests. As before stated, bile pigment never occurs in the fæces in health; it is present when there is catarrh of the small intestine. Blood pigment is usually in the form of hæmatin. As might be expected, ptomaïnes have been obtained from the fæces of certain diseases caused by fungi.

Intestinal Indigestion.

Intestinal indigestion is said to be due to alterations in or diminution of the bile, the pancreatic, or the intestinal secretion. It is almost always attended by gastric indigestion, and may not readily be distinguished from it. *Acute intestinal indigestion* is due to the irritation of food not properly digested in the stomach. It is attended by colic, with flatulency and borborygmi. Some fever may arise, and diarrhœa ensue. In the mild forms the tongue is coated, there is loss of appetite, and some general pains. There is epigastric distress or pain in the right upper quadrant. There is flatulency and constipation. The stools are often clay-colored, or may not be changed. Slight jaundice occurs,

and there is an abundance of lithates in the urine. Accompanying gastric indigestion modifies the symptoms slightly.

The symptoms are more marked and pronounced in *chronic intestinal indigestion*. The *local symptoms* are as follows: Pain which begins from two to six hours after eating. It may be complained of in the region of the liver or below the sternum. It is usually seated in the umbilical region. It is dull, continues two or three hours, or until the next meal is taken. There is some tenderness. With the pain there are tympanites, borborygmi and a sense of fulness in the abdomen; the bowels are constipated, and the stools are hard and dry. The constipation alternates with diarrhœa, and undigested particles of food are passed. The appetite is not lost, but is variable. Hemorrhoids are often present. The *general symptoms* are marked, and are referred to the nervous system and the condition of the blood. There is great depression and hypochondriasis. The patient sleeps badly, suffers from bad dreams and tinnitus aurium; there are spots before the eyes and more or less constant headache. They complain of pain in the back and limbs, and hyperæsthesias or anæsthesias are present. There is inaptitude for mental exertion. Frequently the patient has sudden attacks apparently due to toxins, as sudden fainting, followed by collapse, or there may be vertigo. During these attacks there is great palpitation of the heart, and tachycardia. The extremities are cold and there are cold sweats over the body. Independently of the attacks, the patient is subject to palpitation and some dyspnœa. The urine is always high-colored, acid in reaction, and full of urates and uric acid. Oxalate of lime may be present, and the albuminuria of uric acid occurs. The patient is anæmic; the anæmia develops early. There is some emaciation; in some cases the emaciation is rapid. The complexion is sallow. If there is an abundance of oxalates the patient complains of weight and heaviness about the loins. The stools may contain fat, indicating probable pancreatic disease, if fatty food has been ingested. On the other hand, with loss of appetite, furred tongue, frontal headache, and drowsiness, the stools may be clay-colored and the bowels costive; apparently the bile is at fault.

Acute Intestinal Catarrh.

Cause. Exposure to cold or the direct irritation of mechanical or chemical substances within the intestine. Irritating food that is not digested, or that cannot be digested because of the quantity; spoiled meats and unripe fruits, usually excite an attack. Water saturated with impurities, or the natural characters of which the individual is not accustomed to, may excite an attack. Strangers in a new locality are frequently subject to a diarrhœa until accustomed to the drinking-water, which in the natives does not excite catarrh. Toxic substances, as poisons or drugs, or toxic substances the result of putrefaction, as ptomaïnes, are frequent exciting causes. Extension of inflammation from neighboring structures by infection, as in peritonitis, sets up a catarrh. Local diseases of the intestine, as ileus, intussusception, hernia, and ulcers of all forms, are attended by catarrh of the intestine. In cachectic states of the system, as cancer, anæmia,

and Bright's disease, catarrh occurs. In diseases of the heart and blood-vessels, or of the liver and spleen, on account of which the disturbance of the circulation causes a congestion, catarrhal inflammation occurs. It is of common occurrence in the infectious diseases, and particularly in septicæmia and pyæmia.

Symptoms. Diarrhœa is the chief symptom, varying with the cause and the extent of the catarrhal inflammation. The stools differ in frequency and in color, as has been previously indicated in the various types, and, depending upon the cause, contain undigested matter or worms. Colicky pains about the umbilicus, with borborygmi and frequent desire to go to stool, attend most forms. Each evacuation is preceded by the above symptoms. Fever of the remittent type, with some prostration, attends. The urine is scanty and high-colored. The symptoms vary somewhat with the location of the inflammation, although the exact locality cannot be as distinctly defined as at one time was thought possible. It is nevertheless true that in proctitis there are rectal symptoms of pain with tormina and tenesmus. These are more common than in inflammation which is apparently limited to the small intestine, while in colitis the degree of the rectal symptoms stands between enteritis and proctitis.

The *diagnosis* of acute intestinal catarrh is not difficult. It is more difficult to determine the actual cause. If the attack occurs suddenly following the eating of improper food, or the drinking of impure water, the irritation is probably due to that cause. The cause may be determined by the nature of the fæces. If they contain undigested food the diarrhœa is probably due to indigestion. Catarrh from cold usually follows exposure, and is generally not very severe. To estimate the cause from poisons or drugs the condition of the rest of the intestinal tract must be investigated and other symptoms of the effects of drugs must be inquired for. In arsenical poisoning there is always vomiting, and the discharges are of a choleraic nature. Collapse rapidly ensues. The other symptoms of arsenical poisoning must be inquired for and the history of exposure, if possible, ascertained. The intestinal catarrh due to infectious diseases is attended by the symptoms due to the respective affections, each of which is usually readily recognized. It may be necessary to resort to a bacteriological examination of the fæces. The intestinal catarrh which occurs on account of local disease of the bowel, as hernia, stricture, etc., is preceded or attended by the local symptoms of these diseases. In like manner we judge of the nature of the diarrhœa that occurs in the course of tuberculosis or syphilis, and in the course of organic heart disease or of liver disease. In each instance the possible influence of morbid processes present in other structures must be very carefully estimated.

The Varieties of Acute Intestinal Catarrh. Divisions have been made in accordance with the symptoms which distinguish the various localities of the intestine in which the inflammation is most marked.

Catarrh of the Duodenum. This partakes of the nature of the symptoms of gastro-intestinal catarrh in a mild degree, and is characterized by the occurrence of jaundice due to catarrhal inflammation of the biliary passages.

The Small Intestine. Colicky pains and rumbling are experienced. There is usually gastritis at the same time. The fæces are mixed with mucus. Over the right lower quadrant there is tenderness on pressure.

Cæcum. Pain in the right lower quadrant with tumor, dulness on percussion, with tenderness, are present. (See Typhlitis.)

Colitis. The large intestine is most frequently affected. Pain and tenderness along the course of the bowel. The evacuations contain mucus; there is tenesmus.

The Rectum. Proctitis gives rise frequently to small stools, tenesmus, pain in the left lower quadrant, with tenderness about the anus and spasm of the sphincter. There is considerable mucus and blood in the passages.

Cholera Infantum. This affection occurs in children during the hot season. It is promoted by bad hygienic surroundings and is due to improper milk or food. At first there is catarrhal diarrhœa. This may continue for twenty-four hours, then vomiting and diarrhœa ensue. The stools are liquid and large in amount. At first they may contain milk curds. The vomiting is excited by anything taken into the mouth, or by odors, or by movement of the little patient. The watery discharges are almost constant. They may be preceded by greenish or yellowish-green stools for twenty-four hours. Stools are acid in reaction, and their odor is sour. At first there is colicky pain, but when the watery discharges begin there is only a little tenesmus. The abdomen is at first distended with gas, but soon becomes retracted. The fæces irritate the skin and cause eczema. The rectum may become prolapsed. In a short time, twenty-four hours or even less, collapse ensues. Previous to the collapse the skin is hot and dry, patient restless, the thirst intense, the mouth dry. The body temperature is 103° to 104.° With collapse the extremities become cold, the skin cool. The axillary temperature is lowered and the rectal temperature increased to 105° to 106.° The restlessness continues, the fontanelles become depressed, the eyes sunken, the face pinched, the brows drawn. The urine diminishes in amount or may disappear entirely. Brain symptoms ensue. So-called hydrocephaloid symptoms follow—rolling of the head, strabismus, turning in of the thumbs, and later, convulsions. Stupor followed by coma develops in the fatal cases. If the patient does not die in collapse, marasmus develops; ulceration of the cornea may take place; there is œdema and blood extravasation under the skin. The child emaciates and withers. On account of the weak heart and exhaustion, pulmonary atelectasis or broncho-pneumonia may occur. The age, the season, the presence of the catarrh, with collapse and other symptoms, render the diagnosis easy.

Entero-colitis. In entero-colitis the more intense inflammation succeeds a mild intestinal catarrh. There is increased languor, great fretfulness, and fever. The early catarrh is attended by green acid stools, with lumps of casein. The tongue is furred and moist at first. It soon becomes red and dry; vomiting ensues. The stools are offensive and increase in frequency, and, in addition to the appearance first indicated, contain mucus and blood. Death may take place within the first week on account of exhaustion from the vomiting and diarrhœa. If

the disease is protracted there are great wasting, symptoms of hydrocephalus, skin eruptions, hypostatic pneumonia, and extremely weak, feeble circulation.

Acute Dysentery. The term dysentery is applied to an inflammation of the intestinal tract, chiefly the colon, which is attended by the symptoms of intestinal catarrh in intense degree, characterized by mucus and bloody discharges, with the severe general symptoms of fever and prostration, followed by extreme exhaustion, the occurrence of abscesses in the portal circulation, or of paralysis, arthritis, nephritis, or profound anæmia. It was thought to be an epidemic disease which was mildly contagious. Although of frequent occurrence sporadically, it is common in jails and institutions, in camps, or where people are crowded together, when at the same time hygienic conditions are most unfavorable. It usually occurs in the summer or fall, and is attributed to the drinking of impure water. A form most common in the tropics is called tropical dysentery. Recent investigations have shown that catarrhal dysentery due to the above-mentioned circumstances may occur, and that in addition "tropical" dysentery, although not confined to the tropics, is associated with inflammation and ulceration of the bowel, attended by the amœba dysenteriæ or A. coli.

Catarrhal dysentery may be limited to the simple inflammation of the intestine, or may be followed by ulceration. Its first symptoms are those of intestinal catarrh. There is indigestion, with loss of appetite, perhaps vomiting, and the occurrence of slight diarrhœa. These symptoms may have immediately followed a diarrhœa or a chill may take place after they have continued three or four days. The diarrhœa is attended by pain, at first seated around the umbilicus; it then becomes marked in the course of the colon. The movements are frequent, preceded by constant desire, and attended by extreme tenesmus. The stools, which were first fæcal and fluid, soon become scanty, and consist almost entirely of mucus and blood. The symptoms of local proctitis are intense; there is a sensation of a hot mass in the rectum. There may be strangury, and prolapse of the anus may ensue.

With the active pain and frequent evacuations the skin is hot and dry; there are thirst, nausea, and occasionally vomiting. The temperature continues at about 103°; the pulse is rapid. There are restlessness and weakness; the tongue is red and raw.

If the disease is severe from the start, or the course is unfavorable, stools may contain pure blood, or are dark in color, and may contain shreds of membrane. Pain and tenesmus disappear, and the evacuations become constant or involuntary. Restlessness becomes more aggravated; the extremities become cold; mild delirium sets in. The tossing and restlessness are quite characteristic, and are attended by sighing and some dyspnœa. The pulse is rapid and feeble; the heart sounds are weakened; the tongue becomes dry and brown, the mouth is parched, and thirst is intense; ulcers develop in the mouth and sordes collect around the teeth. The delirium increases to stupor, and from that to coma. The urine, at first high-colored and scanty, becomes bloody and contains albumin and casts. Although the fever is continued during this stage, the extremities become cool, perspiration breaks out over the

forehead, and, instead of typhoid symptoms, the symptoms of collapse may ensue. If the disease is prolonged and the bowels controlled, the symptoms of pyæmia may develop.

The anæmia that ensues is extreme, and wasting is prominent. Convalescence is slow and may be attended by chronic diarrhœa. Before it is established ulcers of the skin may form on various parts of the surface of the body. Arthritis is of common occurrence, and paralysis may occur during convalescence or after an attack has subsided on account of peripheral neuritis. Chronic dysentery may succeed the acute. It is thus seen that the attacks may be of moderate severity or extremely grave; during the course of the latter gangrene of the lower bowel may take place.

Amœbic Dysentery. This differs from catarrhal forms of dysentery in many respects. The onset may be abrupt or gradual, as in the previous form, with symptoms of intestinal catarrh. In most of the cases a frequent and painless diarrhœa follows a period of slight ill health. The diarrhœa alternates with short periods of constipation; the stools are watery and contain mucus, but no blood. The course of the disease is irregular. There may be intermissions and exacerbations of the diarrhœa without obvious cause. It may rapidly pass from one grade to another, or become chronic. One form is the gangrenous, which may scarcely be appreciated by the symptoms until the autopsy shows it to have been present. True relapses are common, and the tendency to chronicity is very great. In the milder cases there are weakness, emaciation and pallor; the expression is dull; the skin is dry and sallow; the tongue pale, flabby, and moist, slightly furred; the abdomen is normal or retracted; the temperature does not rise above 100°, and the pulse ranges from 70 to 90. Sleep is disturbed by frequent evacuations of the bowels. In the grave form the face is drawn, or cyanosed or flushed, the expression anxious; the mind is clear. There are anorexia, intense thirst, and sleeplessness. The abdomen is greatly retracted, and there may be free sweating. The temperature is normal or subnormal; the pulse small and rapid. Progressive anæmia and loss of flesh are prominent and dominate the intestinal symptoms. The skin is dry and harsh, and of a dull greenish-yellow color if the cases are protracted.

The special features of amœbic dysentery are: 1. *The anæmia.* This is due to diminution of the red cells and the hæmoglobin, first, because of the action of the amœbæ upon the red blood-corpuscles, which they destroy; second, the direct loss of blood; and, third, malnutrition. The first is the most predominant. 2. *Diarrhœa* may be the only feature of the disease. It is characterized by great variation in character and frequency in all grades and during different periods of the disease. Intermissions and exacerbations may be observed at any time. The latter begin suddenly, and subside in the same manner. They may last from two to ten days. The intermissions continue from one day to three weeks, during which the fæces are soft, but contain mucus. Councilman and Lafleur have observed this periodicity to be most marked in cases complicated with hepatic abscess.

3. *The Stools.* The stools are extremely variable in accordance with the severity of the ulceration, and also vary in number and character from

day to day in individual cases. In the gangrenous form they number thirty or forty in twenty-four hours at first, then decline, so that toward the end of fatal cases but three or four take place. At first the movements are small and consist of mucus with more or less bright blood and small fæcal masses. As ulceration advances the stools change, they become more copious and watery, fæces are absent, blood is not so frequent. Shreddy masses of grayish or yellow color appear mixed with mucus. If there is sloughing they become greenish or grayish, resembling spinach, or reddish-brown and very liquid or pultaceous. The odor is penetrating and offensive. Shreddy masses of necrotic tissue are discharged. Gray liquid movements, somewhat slimy, contain more pus than the others. Small opaque, or translucent, gelatinous grayish masses, one to three cubic millimetres in diameter, are found in the stools.

In the more moderate types the stools at the outset are like those of gangrenous dysentery if the attack is abrupt. If gradual, the stools are fæcal, liquid, containing mucus and streaks of blood and many of the gelatinous grayish masses. Stools of this character number from four to ten in twenty-four hours; this may continue for weeks. During the exacerbations the stools resemble those of the second period of the gangrenous form. In chronic dysentery there is not so much mucus or blood, except in exacerbations. The stools are of the consistence of thin gruel and have an earthy or dull yellow color. Mucus is persistently present, however, in the intermissions, when the stools are soft and fæcal.

The reaction of dysenteric stools is generally alkaline.

Microscopical Examination. In the mucoid and bloody stools of the acute stage red blood-corpuscles, leucocytes, and large, round, or oval epithelioid cells are seen. The latter are often in groups of three or more. The nucleus is about the size of the red blood-corpuscle, the protoplasm granular. Their outline is sharp. They may be taken for amœbæ. They are non-motile and refract light less strongly. *Cercomonas intestinalis* is present, but bacteria are not abundant. In the later periods the cell elements are less numerous; shreddy and muscular detritus and bacteria are observed, with elastic tissue fibres. Charcot's crystals and phosphates are seen. In chronic dysentery the cell elements are still fewer and amœbæ easily detected.

Amœbæ. Amœbæ are found at all periods of the disease. They vary in different cases and at different periods in proportion to the severity of the intestinal ulceration. They are most abundant in the grayish-yellow gelatinous masses, next in the particles of clear or opaque mucus, and finally in the fluid portions of the stools. In chronic dysentery they are found in all portions. In the intermission of the diarrhœa they may be found in the particles of mucus adherent to the fæces. They disappear as recovery proceeds, although they may be seen after the evacuations become normal. They vary in size and activity. They are more common in the alkaline and neutral stools. They are scarce and are rarely motile in acid stools. In the more active forms red corpuscles are seen.

For their detection the following should be observed: First, the stools

should be passed in a warm bed-pan and kept at a temperature of 30° to 35° C. until an examination is made. Second, this should be done before the stools become acid. Third, the portions of the stools previously mentioned should be selected for examination. They contain amœbæ in greatest abundance. A magnifying power of four hundred diameters is required, although they may be seen with less.

Description of the Amœbæ. When inactive they are round or slightly oblong, are highly refractive, and contain vacuoles of greater or less size. The latter are clear, and vary from small points to one-third of the diameter of the aureola. The ecto- and endosarc may or may not be sharply divided. If they are, the outer is hyaline or homogeneous, the inner is more refractive and contains vacuoles. They are difficult to recognize in this condition, being mistaken for swollen connective-tissue cells. The amœbæ frequently enclose red corpuscles, pus cells, blood pigment, bacilli and micrococci. In a fresh state the nuclei cannot be made out because they resemble vacuoles. The endosarc is not granular, is composed of a dense substance and is highly refracting. When active the movement is characteristic. It may be slow or rapid, and is of two kinds, a progressive movement and one limited to the throwing out of pseudopodia. The movements appear to be rhythmical in some cases, occurring at regular intervals. The movement is sudden and characterized by change in form of the pseudopodia. The ecto- and endosarc are clearly defined usually. The pseudopodia are alkaline and homogeneous, like the ectosarc. The amœba changes its position sometimes by enlargement of the pseudopodia, into which the inner contents of the older part follow. The movements are increased when the examinations are made on the warm stage.

In *catarrhal dysentery* the stools are uniform in character, quantity, and frequency. The onset is sudden, and evacuations consists of bright blood and viscid, clear mucus mixed with fæcal matter. They soon are composed entirely of mucus and a little blood. The mucus is viscid. In a week or ten days the mucus changes and becomes grayish-white in color, is less blood-stained and brown; pultaceous or fluid fæcal matter appears in the stools. As the blood and mucus disappear, formed fæces return. In the prolonged cases the stools are soft, yellowish-brown, or greenish, in addition to the bloody mucoid stools. The frequency is greatest at the onset and progressively diminishes until convalescence is established. The more frequent the evacuations the smaller the size of the stools. The mucoid stools are small, pultaceous, more bulky. On microscopical examination red and white corpuscles, cylindrical epithelial and oval epithelioid cells are seen. The latter are very characteristic, and occur singly or in groups. Bacteria are more common as improvement sets in. In the pultaceous stools the cell elements are scarce. In *diphtheritic dysentery* the stools are watery. They resemble meat washings—evacuations such as are described in cases of *gangrenous dysentery*. They are grayish-green or reddish-brown and very offensive. Mucus is present in small amounts. At first unclotted blood is present, afterward minute dark red clots are seen. Shreddy and finely divided material, gray or reddish-brown in color is present, but there are no sloughs. The stools are not numerous at first, and average from seven to fifteen daily

during the course of the illness. The quantity passed is small. Cylindrical epithelial cells are most abundant on microscopical examination. Red blood-corpuscles and leucocytes are observed, but fibrin constitutes the larger portion of the stool. In all the stools bacteria are present in great numbers.

Other Symptoms of Amœbic Dysentery. Abdominal *pain* is constant; it occurs in the early stages of both forms and in acute exacerbations. As the movements diminish the pain decreases. In the gangrenous form pain also disappears, although the intensity of the process is increasing. In chronic cases the colic is complained of during the exacerbations; during the intervals a dull aching or burning pain is complained of in the upper quadrants. In all cases the pain is cramp-like, boring, or burning in character, and usually precedes and accompanies movements of the bowels. When severe, it is general; but it is usually localized in the lower abdominal zone. Moderate tenderness on pressure can be elicited in most cases along some part of the course of the large bowel. In catarrhal dysentery tenesmus is common; in the amœbic form it is infrequent. A burning sensation in the rectum and at the anus during and after the passage of fæces is generally complained of. Nausea and vomiting occur at the outset, or at irregular intervals, caused by improper food, or on account of complications. Hiccough occurs in the terminal stages.

Fever. In amœbic dysentery fever is not a prominent feature, although there is usually a moderate rise in temperature. In the gangrenous form it is normal, or may be subnormal for days. Chronic dysentery is afebrile. In exacerbations of diarrhœa slight fever may occur. Complications cause a higher temperature If fever is present it may be remittent or intermittent in character, or if the illness is prolonged at any time, first continuous, then remittent and then intermittent. If the latter, the usual morning fall is observed, although an inverse temperature may be present. Rigors occur with the complications. Sweating is observed, with subnormal temperature, in the gangrenous form. In cases of abscess the fever is intermittent or remittent.

In chronic dysentery the skin is excessively dry. The circulation and respiration are influenced by the pyrexia. Anæmia is pronounced. When exhaustion ensues the pulse becomes more feeble, compressible and rapid. The urine is albuminous, and often contains casts. In the gangrenous form there may be retention of urine.

The complications of amœbic dysentery are: 1. Hepatic abscess, or hepato-pulmonary abscess. 2. Peritonitis. 3. Hemorrhage from the bowels.

Abscess may develop in all forms and at any period of the disease. The time of the disease at which it occurs cannot be determined definitely. In the subacute cases it is liable to develop from the fourth to the twelfth week. The abscess may develop on the convex surface of the right lobe of the liver near the coronary ligament. In these cases the lung also becomes involved. Councilman and Lafleur suggest that infection takes place by the peritoneum. (See Abscess of the Liver.)

While the symptoms of abscess of the liver will be treated of under the section devoted to liver disease, it is important to note that hepatic

symptoms may occur in cases in which, on account of the mildness of the disease, the local bowel trouble may be overlooked entirely. If the association of hepatic pain with fever and discharge of mucus from the bowels is observed, it is barely possible, even if an examination of the fæces cannot be made, that an hepatic abscess is present. If in addition, cough and expectoration occur, involvement of the lungs is possible. The character of the expectoration points conclusively to the nature of the lung complication. After a period of dry, hacking cough, sudden expectoration of muco-purulent or bloody sputum takes place. It is of a dirty red or brownish puriform color. From this time on this material is expectorated in varying quantities after a paroxysm of coughing. The expectoration is diffluent, tenacious, and frothy. It varies in color from bright red to russet brown; it may be bile-stained. The sputa are alkaline; the odor is not putrid. At a later period they become more purulent, and contain less blood. The sputum separates into three layers: an upper frothy layer, a middle layer of turbid fluid, a thin layer of muco-pus below. Large amounts may be coughed up in twenty-four hours; the sputa contain, on examination, blood-corpuscles, leucocytes, round alveolar epithelial cells and polyhedral, fatty degenerated cells which look like liver cells. Elastic tissue fibres from the lungs are found with crystals of hæmatoidin and tyrosin, and Charcot's crystals. Bacteria are present. Amœbæ are constantly present. They vary in size and activity, but are larger than those seen in the stools. The sputum should be kept warm and examined for them as soon as possible.

Peritonitis. Peritonitis from perforation is not a common complication of amœbic dysentery, but occasionally takes place in the gangrenous form. Peritonitis without perforation may occur. The symptoms do not differ from peritonitis under other circumstances. *Hemorrhage* from the bowel occurs and may be sufficiently profuse to cause death. Other complications which have been described under catarrhal and croupous dysentery are likely to occur in this affection.

The Diagnosis. The diagnosis of this form of dysentery is made absolute by finding the amœbæ in the stools. The history and the course of the illness must also be taken into consideration, the characteristics of which have been previously detailed. The irregularity, and the intermittency of the diarrhœa, the infrequency of tenesmus, the moderate fever, the reaction of the stools, and their comparative freedom from bacteria, are further corroborative points.

Cholera Morbus. The attack is characterized by sudden vomiting, followed in a short time by purging. The vomiting may be preceded by pain, or both may occur at the same time. At first the pain is seated in the epigastrium and subsequently about the navel. It is very severe and paroxysmal in character, compelling the patient to double up if lying in bed. A cold perspiration breaks out on the forehead, the extremities become cold, the face is anxious, the pulse becomes rapid. At first the patient vomits undigested food, then watery, greenish-colored fluid. The latter is bitter. Purging sets in at once, or within an hour. The bowel movements follow an attack of pain. The first passage is fæcal, and may contain undigested food, the

subsequent passages are watery and profuse. There are severe attacks of burning and tenesmus; the abdomen is tender around the navel and in the epigastrium. After an evacuation there is slight relief, but soon another paroxysm of pain comes on. The vomiting is excessive, and retching may be present in the intervals. Ice, or water, or anything taken into the stomach excites pain and causes the vomiting. The attack subsides in twelve to twenty-four hours, followed by exhaustion. In rare cases collapse ensues, and in others it is followed by gastro-intestinal catarrh.

Cholera Nostras. This affection occurs in epidemics in hot weather. The symptoms are those of severe gastro-enteritis. There is sudden vomiting and diarrhœa. It usually begins in the night. The vomiting is not different from that of cholera morbus. The watery and brownish-colored stools become colorless and have the appearance of rice water. Pain attends the attack, rapid prostration ensues, the extremities become cold, and collapse takes place. With the collapse there are cramps in the legs. Other muscles of the body may become cramped. The disease occurs in epidemics during the hot season, and may be mistaken for cholera. It can only be distinguished from the milder forms of cholera which precede the occurrence of the epidemic by the absence of the comma bacillus. The bacillus of cholera nostras is found in the stools. (See Fæces.)

Chronic Intestinal Catarrh. It usually follows an acute attack, or may be chronic from the start. It arises secondarily to portal congestion in disease of the liver or spleen, to chronic disease of the heart, or of the lungs. It occurs in malaria and in the scorbutic cachexia.

The symptom is diarrhœa alternating with constipation, or diarrhœa alone occurs. Stools may contain undigested food, or pus and mucus and blood in small amounts. Diarrhœa may be present in the morning only, under these circumstances. If the fæces are examined, the eggs of parasites, or infusoria may be found. The local abdominal symptoms of rumbling, flatulency, and tormina are present. There are reflex symptoms of cardiac palpitation and dyspnœa (asthma). Rush of blood to the head may occur. Often these symptoms are relieved by the passage of flatus. Chronic catarrhal gastritis usually accompanies the intestinal catarrh. The general symptoms of *anœmia*, *emaciation* and *neurasthenia* are present. Hemorrhoids are common.

Ulceration of the Intestines.

Duodenal Ulcer. Ulcer of the duodenum usually occurs in young subjects in whom there are symptoms of chlorosis or anæmia. The causes of gastric ulcer usually exist. It may follow boils, erysipelas, or pemphigus, and differs in one etiological respect from ulcer of the stomach in that it occurs most frequently in the male sex. The symptoms are obscure, and may be wanting entirely, the patient probably complaining only of intestinal indigestion. In other cases they are like those of gastric ulcer. In typical cases the symptoms are those of pain situated below the xiphoid or to the right of the median line in the region of the pylorus. The pain occurs after eating, and may be relieved by

vomiting. There is localized tenderness on pressure. Hemorrhage may take place from the stomach, or blood be found in the stools alone. It differs from gastric ulcer only in the possible difference in location of the pain, the occurrence of intestinal indigestion and gastric hemorrhage, and the fact that the pain continues several hours after eating.

General Ulceration. Ulceration of the intestine may be due to a specific infection, and hence symptomatic of typhoid fever, syphilis, and tuberculosis. It is always present in the former, and of frequent occurrence in the latter. Follicular ulceration occurs in entero-colitis in children. Ulcers due to the pressure of fæces occur in typhlitis and chronic constipation. The sacculi of the colon become filled with scybalous masses, the pressure of which produces ulcers. Tenderness is experienced along the course of the colon, particularly on palpation of the fæcal masses which may be felt through the abdominal wall. A non-specific chronic ulcerative colitis is the form that succeeds the diarrhœas which occur during camp life, or that are set up in communities where people live closely under bad hygienic circumstances. It is the form that attends scurvy, and is frequently seen in chronic Bright's disease. It may be succeeded by dilatation of the colon, by hypertrophy of the muscular walls, or by contraction of the bowel. The persistent diarrhœa leads to profound emaciation, extreme prostration, sallow complexion, with markedly impaired nutrition of the skin. Such forms of diarrhœa were seen during the late war, particularly in soldiers held in captivity. The diarrhœa may first be of a lienteric character, and later alternate with constipation. Stools contain blood and mucus. Most of the pensions given to soldiers at the present time are given because of this disease.

Ulcers of the intestinal tract may occur from other causes and diarrhœa be the prominent symptom. They may be due to cancer; the malignant nodules may ulcerate within the lumen of the bowel. The bowel may be perforated from the exterior, on account of suppuration somewhere along its course, as in appendicitis, pancreatitis, or tuberculous peritonitis. The *symptoms* of intestinal ulcer are those of diarrhœa. Ulceration, however, may be present without any symptoms whatsoever, particularly if the small intestine is affected. One or two small ulcers, on the other hand, in the lower portion of the colon may set up continuous diarrhœa. The *stools* are composed of fæces, mucus, pus, shreds of tissue, and blood. If pus is discharged in large amounts an abscess has probably opened into the bowel. Moderate discharge of pus usually follows ulcers in the colon. Pus may be present in cancer. *Hemorrhage* is of frequent occurrence, and is an important diagnostic symptom, especially if profuse and occurring without symptoms of obstruction, of gastric ulcer, or of hemorrhoids. The fragments of tissue found in the stools may point to the nature of the process. Large amounts attend the dysenteric process. The fragments may be composed of the mucosa, connective tissue, and the muscular coat. *Pain* occurs in many of the cases. It may be general and colicky, or circumscribed in cases of ulcer of the colon. Perforation of the intestine is followed by localized or general peritonitis. The occurrence of the latter depends largely upon

the situation and the rapidity of the ulceration. If the perforation is in the posterior wall of the colon a circumscribed abscess may develop. When it is situated in the upper zone the pus may accumulate underneath the diaphragm, or in the lesser peritoneal cavity. The signs of pyo-pneumothorax subphrenicus occur when the latter accident takes place, as both pus and air accumulate in the abscess cavity. In such instances the ulceration usually takes place at the splenic flexure. Perforation of an ulcer of the cæcum may simulate appendicitis.

Intestinal Obstruction.

Intestinal obstruction may be acute or chronic, depending upon the cause of the disease. *Acute intestinal obstruction* is due, first, to constriction by bands or strangulation of the bowel through apertures; second, to volvulus of the colon; third, to acute intussusception. In the first instance the type of the obstruction is seen in strangulated hernia, but similar *strangulations* occur in apertures within the peritoneal cavity. Thus, loops of the intestine are caught and constricted in the duodeno-jejunal fossa, the so-called Trites' retro-peritoneal hernia, or in the foramina of Winslow, also known as inter-sigmoid hernia; finally, diaphragmatic hernia, in which protrusions of the intestine into the diaphragm along with other abdominal viscera may take place. The above-mentioned forms of hernia may exist without symptoms, or may, from some unknown cause, lead to constriction or twisting of the loop of the intestine, with the occurrence of acute obstruction. Abnormal lacerations in the omentum may give rise to internal constrictions. Internal constrictions, however, take place, most commonly in the regions of hernias, on account of the gut being constricted by dense fibrous adhesion; or about the uterus or Fallopian tubes, which had previously been the seat of inflammation. The constricting bands that follow the local peritonitis may gradually occlude the gut, or be in such position that the latter becomes twisted about it. In other forms of peritonitis similar constricting bands may form, which are liable to produce this accident. Disease about the vermiform appendix, with secondary adhesions, has been observed to cause constriction. A frequent form of intestinal obstruction is due to the tangling of the intestine in the fœtal remains of the omphalo-mesenteric duct, which, as well as Meckel's diverticulum, is situated a short distance above the ileo-cæcal valve.

Volvulus is a form of obstruction due to twisting or knotting of the intestine. The condition is not common. It occurs most frequently at the sigmoid flexure of the colon. The mesentery of the latter is often congenitally narrowed, on account of which the colon is unduly dragged upon, and, if filled with masses of fæces, cannot restore itself; the twisting becomes permanent, and obstruction takes place. Peristalsis is set up and other portions of the intestine wind about the pedicle of the loops so as to form a regular knot. Abnormal peristalsis on account of diarrhœa often precedes the appearance of the obstruction. *External injury* is said also to give rise to the formation of an obstruction.

Intussusception, as a cause of intestinal obstruction, occurs most

frequently in children, and is due to a portion of the bowel being pushed into the lumen of that which lies next below it. A circumscribed portion of the intestine may be paralyzed. In the portion above, the peristaltic action continues and the energetic movements push it into the paralyzed part. Intussusception is found frequently after death in the bodies of children dying from exhaustion. In such cases it occurs just before death. Intussusception also occurs when intestinal polypi drag one portion of the bowel into the lower portion. Large portions of the intestine may be involved. The invagination usually takes place at the lower portion of the ileum, or in the cæcum; sometimes the invaginated portion may reach the rectum and project externally. Intense inflammation and adhesion are set up. The internal portion becomes gangreneous on account of constriction of the afferent vessels. This portion may slough and pass with the dejections, followed by spontaneous cure.

Intestinal obstruction, to view it from another standpoint, may be due to (*a*) diseases outside of the intestines; (*b*) to disease in the intestinal walls; (*c*) to accumulation within the intestine.

The obstruction takes place under the same circumstances as obstruction in other channels.

A. *Diseases Outside of the Intestines.* 1. Pressure of *tumors*, chiefly ovarian tumors, uterine tumors, tumors of the omentum, and pelvic abscess, or abscess about the cæcum. The symptoms of obstruction develop gradually, although rarely they may take place suddenly, especially if aided by the accidental occurrence of fæcal impaction.

2. *Constricting bands*, hernial openings, the remains of fœtal structures, cause constriction of the intestine. In this class of cases there is usually pain, and the history preceding the obstruction is that of peritonitis, general or local, of old hernia, of appendicitis, of pyosalpinx, or of inflammation about the gall-bladder and gall-ducts. If the constriction is due to protrusion into hernial openings, the onset is usually sudden and without previous symptoms.

3. *Peritonitis* is the most common cause of intestinal obstruction. It may be due to overdistention by gas and paresis of the bowel, or to pressure by external exudation.

4. Knots and twists of the intestines, usually seated about the sigmoid flexure, causing *volvulus*, are a common cause of constriction.

B. *Disease of the Intestinal Walls.* 1. Invagination, or intussusception, in which one portion of the bowel is drawn into the other. It usually occurs in children and is seated in the right lower quadrant in the neighborhood of the cæcum. The attack is acute, although the affection may continue over a long period of time.

2. Cancer of the intestine in its course generally leads to stricture and obstruction.

3. The healing of ulcers, which are syphilitic in the larger number of cases, rarely tuberculous, will lead to stricture. The obstruction takes place gradually in this class of cases. It is seated, in the larger number of instances in the rectum or sigmoid flexure of the colon.

C. *Accumulations Within the Intestines.* 1. Fæces. The obstruction takes place gradually, occurs in weak and debilitated people

in the course of constipation, and follows the constipation of acute disease.

2. Accumulations of improper food or foreign materials. The seeds of fruits or the husks of grain accumulate and cause obstruction. Magnesia, iron, and other articles taken as medicines, from their accumulation lead to obstruction of the intestine. In these instances the obstruction takes place gradually.

3. Impaction of gall-stone within the intestine is followed by acute obstruction.

It will be observed in the detailed list of causes that obstruction may be acute or chronic. Complete acute obstruction may set in in the course of chronic obstruction due to stricture of the bowel, and occlusion due to external pressure or to accumulations within the bowel.

In a case in which the symptoms of intestinal obstruction occur it is important to ascertain, first, the duration of the obstruction and mode of onset; second, the possible cause of the obstruction; third, the seat of the obstruction. The *symptoms* of intestinal obstruction depend upon the nature of the obstruction and the rapidity with which it has taken place. *Constipation.* In all forms of obstruction the one symptom is stoppage of the intestinal contents. When this takes place suddenly, and at the same time there is a local injury to the bowel, the symptoms, both local and general, are most pronounced and alarming. On account of the obstruction there is acute constipation, without the escape of flatus. *Pain.* For the same reason there is pain at the seat of obstruction. This occurs suddenly, and is intense and lancinating in character, radiating from the point of obstruction. Over the part that is painful there is tenderness. *Tumor.* In many instances a tumor can be outlined due to single loops of intestine, thickened walls, or abnormal contents. This is particularly the case in the obstruction of invagination and the obstruction due to volvulus. *Peristalsis.* The obstruction further causes *increased peristalsis.* This takes place above the point of constriction. Sometimes the movements of the intestine can be seen through the abdominal walls. *Meteorism.* The obstruction causes accumulation of gas above the point giving rise to meteorism. If the obstruction is low down, the distention and meteorismus are general. If high up, as in the small intestine, on account of constriction by Meckel's diverticulum or internal hernia, the meteorism is in the upper part of the abdomen, and may be limited in extent, or dilatation of the stomach alone may be present. *Vomiting.* Vomiting soon occurs in acute intestinal obstruction due to decomposition of intestinal contents, to irritation of the stomach by the intestinal contents, to a trauma of the peritoneum at the seat of the obstruction, or, finally, to the occurrence of peritonitis. At first the contents of the stomach are ejected, then watery fluid, bile-tinged or largely made up of bile, and later fæculent matter. Although of fæcal odor, true stercoraceous vomiting occurs later in the course of acute intestinal obstruction. It must not be forgotten that any obstruction of the intestine may accumulate with extreme rapidity, so that fæcal vomiting may occur within two hours of the commencement of an obstruction. It is recognized by the odor of the matter vomited and by its color. It is a grave symptom, indicating complete

obstruction of the intestine. *Eructations of gas* are frequent. The *general symptoms* are those of *shock* in its most pronounced form. Very rapidly the *abdominal facies* previously described develops. In a few instances, as in invagination, there may be *fever*, yet at once, or very soon in its course, the temperature falls to normal or subnormal, or remains at this point if it has not risen. The extremities are cold, the features pinched, the eyes sunken, the expression anxious. The pain causes the patient to double up in bed. The pulse becomes rapid, weak, thready in character, respirations proportionately hurried. The mind remains clear until the supervention of peritonitis and septicæmia.

Chronic Obstruction. The symptoms are those of *chronic constipation*, with local symptoms due to the cause of the obstruction. The bowels are moved infrequently, and then in small amounts. In obstruction due to stricture from cancer, or cicatricial closure, the fæces are ribbon-shaped. Reference must again be made to the occurrence of diarrhœa, or the passage of small scybalous masses, on account of impaction of fæces. In chronic obstruction the general symptoms are those of inanition, with the nervous train of symptoms that have been described in constipation; while the local symptoms depend upon the cause. When the local symptoms are due to the pressure of a tumor, or accumulation of pus or fluid within the abdomen, there is a history of the occurrence of local disease, on account of which the tumor developed; such history is obtained in fibroids or ovarian tumor, or in previous inflammation, which was followed by the occurrence of a tumor about the locality of the inflammation, as the pelvis or the appendix.

If the obstruction is due to cancer of the intestine, the symptoms of that affection are present. A tumor can be made out at some situation in the course of the bowel. If the cancer is seated in the rectum there are tormina and tenesmus, and the discharge of blood and scybalous masses. Local examination reveals the presence of a malignant mass. Obstruction due to stricture from the healing of an ulcer is seated in the rectum or sigmoid flexure of the colon. Pain and a sense of obstruction are referred to that locality. A history of syphilis can be obtained, and frequently the rectal tube, or finger, will detect the stricture. In both instances just mentioned there is a history of imperfect, irregular action of the bowels from time to time, with intervals of comparative comfort. These symptoms precede the constipation. When fæces accumulate in the colon the larger accumulations take place in the sigmoid flexure and in the cæcum. Fæcal tumors, described under Constipation, are felt through the abdominal walls. Obstruction from fæcal accumulation is preceded by a history of constipation (*q. v.*). The accumulations generally can easily be discerned. It must not be forgotten that chronic intestinal obstruction may at any time become acute.

Chronic intestinal obstruction always occurs in *adults*. The *onset* is gradual. Of the symptoms that attend obstruction of this form, *pain* is intermittent, and if there is fæcal accumulation, is not very prominent. *Vomiting* occurs late in the disease, is small in amount, and generally is not a prominent factor. Obstruction to the passage of fæces may be constant or alternate with *diarrhœa*. In fæcal accumulation it becomes complete, although spurious diarrhœa may attend it. The dis-

charges may be bloody, which points to cancer. *Tenesmus* is present in stricture low down in the large bowel. *Meteorism* is not marked when obstruction is high up, as in acute obstruction. When the obstruction is in the large intestine it may be extreme, and in fæcal obstruction gradually increases as the obstruction becomes more marked.

The forms of chronic obstruction that are attended by tumor have been mentioned previously. Coils of intestine in peristaltic movement are seen only in cases in which there is marked emaciation.

Differential Diagnosis. When the symptoms of acute obstruction are present, it is essential to distinguish the form by ascertaining the *nature* of the obstruction, and determining, if possible, its *seat.* Varying factors must be considered in order to estimate the cause of the obstruction. Of these, first, the *age.* Obstruction from intussusception occurs early in life; from bands or through apertures, in adult life, usually prior to forty years of age; in volvulus, between forty and sixty. Obstruction due to a gall-stone occurs during the middle or later period of life—always after forty.

Previous History. In obstruction by bands of adhesion there is a history of peritonitis, or, as Treves points out, previous attacks of obstruction more or less marked. In volvulus the patient has been subject to constipation prior to the attack, and in intussusception there has been no previous history, unless polypus was present, causing dragging, colicky pains, and occasional discharge of blood.

Symptoms. The symptoms of the various forms of acute obstruction vary somewhat. *Pain* in strangulation, from bands or hernia, is severe and paroxysmal in character, attended by collapse. It is also early in volvulus, though not as severe as in the former, and occurs at long intervals, becoming constant with exacerbations. In acute intussusception the pain occurs early, and is steady. It increases, and then may suddenly subside. At first it is paroxysmal, attending discharge of blood and mucus from the bowels. *Local tenderness* in the first group of cases occurs late. In volvulus it occurs early, and may be noted over distended coils. In intussusception it is usually common about a sausage-shaped tumor. *Vomiting* is marked and occurs in strangulation, soon becomes fæculent, and increases the severity of the paroxysms of pain. In volvulus it does not come on so quickly, but is severe and constant when it takes place. The relaxation that attends vomiting often affords relief to the obstruction. In intussusception it does not occur as early as in the other forms, and is not so severe. It becomes fæculent in a small number of cases only.

Constipation is continuous in all cases except intussusception. In the latter there is some constipation, but it is not absolute; diarrhœa is not uncommon, and discharge of blood in the stools occurs in 80 per cent. of the cases, according to Treves. *Prostration* is severe in all cases, although probably not so marked in volvulus. Because of its close proximity to the rectum, *tenesmus* occurs in volvulus, and is of frequent occurrence in intussusception, often beginning early in the attack.

Physical Signs. On palpation of the abdominal wall it is noted to

be soft and flaccid in most of the cases, unless peritonitis has ensued. This occurs early in volvulus, and rigidity is likewise marked. In a large number of cases a *tumor* can be made out in intussusception only. It is seated in the lower right quadrant of the abdomen. Early in the attack it is oblong and of sausage shape. When peritonitis ensues it disappears on account of the tympany. A portion of the gut may protrude at the anus, or be felt on rectal examination. *Meteorism* occurs about the third day in a strangulation; it occurs early, is very rapid and pronounced in volvulus, and is absent in intussusception, unless constipation takes place.

The Seat of Obstruction. The seat of obstruction is in a measure indicated by (1) the location of the pain, (2) the character of the swelling, (3) the character of the stools, (4) the degree of meteorism, and (5) the results of a rectal examination. In obstruction which occurs high up there is but little meteorism, the tumors are usually not detected, and pain is seated about the umbilicus or the upper quadrants of the abdomen. Obstruction that takes place at the ileo-cæcal valve may be indicated by a tumor in the lower right quadrant over the region of the valve or just above it. It is usually at this point that invagination takes place, and hence we may look for tumor in this situation. On the other hand, volvulus of the colon, or stricture of the rectum, the obstruction being low down, is attended by much meteorism and pain in the left lower quadrant of the abdomen. In volvulus tumor may be detected in this position, and there is much meteorism. The position of the obstruction is sometimes indicated by the seat of *peristalsis*. This may be seen to stop at a given point, which usually indicates the position of the obstruction. In general it may be said, the more severe and rapid the symptoms the more likelihood that the obstruction is in the small intestine.

The Urine. The position of the tumor, it is said, can be ascertained by changes in the urine. When the obstruction is in the small intestine, indican is much increased from the decomposition of albuminous substances and other products of putrefaction. In this location the urine may be suppressed. In stenosis of the large intestine it is not increased unless there should be cancer.

Intussusception or invagination occurs most frequently in children prior to the tenth year. It is characterized by severe colic, and pain in the abdomen, first complained of about the navel. The severity increases in paroxysms, and only lessens if complete strangulation has taken place. With the onset of the pain there are one or two movements of the bowels which contain mucus and blood. After this there may be constipation, or the stools continue to be loose, and are as frequent as fifteen or twenty in a day. Sometimes they are quite bloody, and almost always there is some tenesmus. In a short time after the attack vomiting commences. It may be constant or occur only after taking food. At first the abdomen is soft, but tender on pressure. A sausage-like tumor may be felt on the right side below the transverse umbilical line. On inspection of the rectum a portion of the intestine may be seen, dark and gangrenous in appearance, or it may be felt by palpation. If there is much tenesmus, the anus often remains open. In rare cases the bowel may slip back and the symptoms subside spontaneously.

On the other hand, peritonitis may rapidly ensue, with high fever, followed by collapse and death.

Diagnosis. It must be distinguished from the *entero-colitis* of childhood, or the proctitis due to a polypus. In entero-colitis there is no tumor, and the collapse and prostration do not occur so early, and are not so rapid. There is greater likelihood of a number of the stools being greenish, like spinach. In a polypus of the rectum the symptoms are local, the child is worn out and restless, but great abdominal tenderness, and the tumor, meteorism, vomiting, and collapse do not take place. The rectum must be examined.

Intussusception must be distinguished from *peritonitis* in which symptoms of stenosis of the bowel from ileus paralytica may be present. The history and sequence of events must be watched carefully. Often the commencement of the affection about hollow viscera which have previously been the seat of disease, or its onset with sudden perforation, will point to the nature of the affection. In *peritonitis* there is no active peristalsis; there is general distention of the abdomen, with general tenderness; the urine is diminished, but does not contain indican in excess. Collapse ensues rapidly. Signs of effusion within the abdomen may appear.

Course of Hernia and Volvulus. Obstruction due to these conditions occurs in adults after the fortieth year of age, usually in both sexes. In stricture from the pressure of bands there has usually been a history of previous attacks of peritonitis or of inflammation of the structures in relation to the peritoneum. The attack begins suddenly, and the symptoms may from the start be most pronounced. They are the typical symptoms of intestinal obstruction previously described. The local tenderness, however, may not be present as early as in other forms of obstruction. It is quite characteristic, however, to be unable to find a tumor or positive local cause for the obstruction, also not to have meteorismus. This is due to the fact that the obstruction is usually high up in the intestinal tract.

Volvulus. Volvulus occurs most frequently in males. It occurs late in life, and is usually preceded by a history of constipation. Premonitory symptoms may have been present for a few days, but the symptoms of obstruction take place suddenly. They are the symptoms of acute obstruction, but as the lesion is in the lower portion of the bowel meteorismus is present to a marked degree, and rectal symptoms are found. Tenesmus is present in a small proportion of the cases. Peritonitis is likely to set in early with increase in the temperature, increase in the tenderness of the abdomen, and more pronounced symptoms of collapse.

Diagnosis of Intestinal Obstruction. Intestinal obstruction must be distinguished from peritonitis and appendicitis. This is sometimes very difficult. Careful attention must be paid to the evolution of the case and the history of previous abdominal disease, or of lesions on account of which, on the one hand, peritonitis may occur, or on the other, obstruction of the bowel. In peritonitis the attack follows disease in the uterine appendages, the vermiform appendix, or the gall-bladder; or from perforation in some portion of the gastro-

intestinal tract. *Fever* usually attends the inflammation, with or without a chill. *Vomiting* will probably occur at the onset, and then subside until the peritonitis becomes general. The first paroxysms of vomiting are apparently due to shock. The vomiting that occurs rarely becomes fæculent. As the peritonitis advances it is not an active action, but instead a passive one; a simple constant regurgitation of a large amount of fluid, greenish or grayish-yellow, or watery, takes place. It pours into the mouth, and is simply discharged without the occurrence of retching. The abdomen is swollen and *tympanitic*. The symptoms due to excessive tympany are more marked than in intestinal obstruction. The diaphragm is interfered with, breathing is hurried. It is tender on pressure and is the seat of general *pain*. The general pain and tenderness, however, can usually be found to be more marked at some one of the situations which is the primary focus of the the disease. Further, on local examination, in these positions fulness or undue prominence or *swelling* may be observed. *On palpation* over the point of origin there may be localized *œdema*. The symptoms of collapse do not differ from those of intestinal obstruction in marked degree, although the peculiar appearance of the face and other nervous features occur more rapidly in peritonitis than in obstruction. It must be remembered that peritonitis in a large majority of cases attends obstruction.

In *appendicitis* the symptoms are somewhat like those of intestinal obstruction. There may be constipation, and the occurrence of vomiting. The former is not pronounced, and can usually be relieved. Vomiting subsides after the first twenty-four hours, unless peritonitis supervenes; it is never stercoraceous. The local physical signs are characteristic. In appendicitis there is fixed tenderness on pressure at McBurney's point. Some swelling can almost always be observed. On light or deep percussion there is change in the note as compared with the other side. Fluctuation can often be detected in from two to four or five days. Both the tumor and fluctuation can be detected by bimanual examination of the abdomen and flank. Examination by the rectum may reveal a tumor at the brim of the pelvis on the right side. Fever attends the attack throughout. When peritonitis supervenes there is rigidity of the entire abdomen, which at first was localized to the right lower quadrant.

Intestinal obstruction must not be confounded with *enteritis*. In all forms there is diarrhœa, in many vomiting. Pain of a colicky nature, spreading from the neighborhood of the umbilicus, is marked whenever obstruction to the passage of fæces or gas takes place. Vomiting is not stercoraceous, and the general symptoms, collapse, etc., do not occur. Acute *hæmorrhagic pancreatitis* is also attended by symptoms similar to those of intestinal obstruction. There is sudden severe pain in the upper half of the abdomen, with vomiting, and the rapid develowment of collapse; there may be constipation; the situation of the pain is of some significance. Vomiting never becomes stercoraceous, flatus can usually be passed and the bowels opened by an enema. Meteorismus does not take place. If the symptoms are not too severe there may be increased dulness, and possibly a tumor on deep palpa-

tion in the left upper quadrant of the abdomen along the margins of the ribs, which should be dull on percussion, or, on account of its relation to the stomach, give a dull tympanitic note. The symptoms of internal hemorrhage are present, pallor of the face and extremities, syncope, and in addition prostration and other symptoms of collapse.

Appendicitis.

This is by far the most important affection of the intestinal tract. It is of frequent occurrence compared with intestinal obstruction, and if recognized is amenable to relief in a very large percentage of the cases; whereas intestinal obstruction is frequently fatal. We see twenty-five cases, at least, of appendicitis in all forms to one case of any form of obstruction. Its importance, therefore, is readily recognized. Appendicitis occurs most frequently in the young—in the large proportion of cases under thirty. I have seen it as early as two years of age, although from the fifteenth to the thirtieth year it is more frequent than at any other period. The symptoms vary, but clinically may be divided into those of appendicitis without perforation and appendicitis with perforation. Appendicitis without perforation is characterized by relapses, and is known also as *recurring appendicitis.*

Appendicitis without Perforation. There are probably cases of catarrhal appendicitis, although I am not prepared to say that catarrhal inflammation of the appendix gives rise to marked local symptoms, for in cases on the post-mortem table in which the lesions of catarrh were found, there had not been any symptoms during life due either to intestinal catarrh, or to any symptoms pointing to appendicitis in any form. Moreover, many cases in which the attacks of appendicitis had been slight finally come to an attack with perforation. In these cases of the lighter attacks, if operative measures are resorted to during the interval, they are always found to contain a fluid loaded with micro organisms which are capable of causing purulent inflammation, as the staphylococcus or streptococcus. Clinically, therefore, all forms of appendicitis should be considered due to purulent inflammation, with, on the one hand, escape of the contents into the bowel, and natural relief of the symptoms; or, on the other, complete obstruction with perforation. In recurring appendicitis in which the appendix was removed during the interval, I have always found pus or a mucopurulent material which was charged with streptococci or staphylococci, as well as the bacillus coli communis, natural to the intestinal canal in this region.

Symptoms of the Attack. After exposure to cold rarely, frequently after an indiscretion in diet, the patient is seized with pain, referred to the right lower quadrant of the abdomen. It is paroxysmal in character, increasing in intensity, and may be complained of as colicky. The pain is usually such as to require the patient to take to bed and attempt to secure relief by local applications. The severity of the pain may be such as to require the treatment previously noted, or only of such degree that the patient pays but little attention to it. He even may go about his business during the time and seek professional

advice at the office of a physician. It is this class of cases that are attributed to ordinary cholera morbus or intestinal indigestion. It may be moderately severe only, particularly if there is diarrhœa. With the onset of the pain vomiting usually occurs. At the same time the bowels may be opened, or they may be confined. Vomiting may not occur if there is diarrhœa. Vomiting is usually attended by some nausea, although this is not marked. The vomiting is complete, there is no retching. It occurs at intervals, between which there is comparative comfort. The contents of the stomach are ejected, and then mucus. If the patients are to get well, vomiting does not return unless excited by food. If peritonitis supervenes in the course of three or four days vomiting returns. The patient lies on his back with the right leg flexed.

Even with a mild degree of pain, the skin is hot and the temperature slightly raised. In the cases in which the pain is more severe the general reaction is greater. The temperature rises rapidly to 102° to 103°. The skin is hot and dry, the face flushed. The pulse in a young adult rises to 90 and 95. It is full and strong. On account of the pain there is some restlessness. In some cases the patient complains more of the fever than of the pain after the first severity of it has subsided. The tongue is coated; appetite is lost.

On *physical examination* the area which was the seat of pain is tender. When examined with the tip of the finger pressing firmly, a point of more marked tenderness can usually be found on a line midway between the anterior superior spine of the ilium and the umbilicus. It is known as McBurney's point, and is most characteristic. It is due to tenderness elicited over the site of the appendix. On *inspection* the affected area is slightly or may be considerable enlarged. Comparison must be made with the opposite side. It will be seen that the usual depression in front of the anterior spine, or the cavity toward the loin, is not as deep as on the opposite side. In front the surface may be even with the plane of the ilium. On *palpation*, in addition to tenderness and pain at the point previously indicated, fulness and enlargement can be distinguished. There is resistance to pressure and more or less rigidity of the abdominal muscles. On careful measurement the semi-circumference will be found in most instances to be larger than the semi-circumference of the opposite side. When bimanual palpation is performed, the left hand being placed in the loin behind and the right over the abdominal surface, resistance, induration, and rigidity can more easily be detected. On *percussion* there is change in the note compared with that of the opposite side, and change in the percussion note during the course of the disease. This is particularly the case if the symptoms go on to perforation. On careful deep percussion a dull tympanitic tone is elicited, or a distinct area of dulness can be mapped out, but in some instances the distended cæcum yields tympany which is greater than on the opposite side.

The *pain* is usually referred to the region above mentioned. It may, however, be referred to the bladder or genitals, and be attended with vesical tenesmus and frequent micturition. The character of the

pain and the bladder symptoms are such as to simulate an attack of renal colic, with the passage of sand. On account of the locality of the pain it may be attributed to the Fallopian tube or ovary and thought to be due either to pain on account of disease of these organs or to dysmenorrhœa. It is not likely to be mistaken for the pain of dysmenorrhœa if the patient is subject to pain at the usual monthly period. If, however, the physiological and pathological affection should take place at the same time or the latter occur about the time of the monthly period a mistake in diagnosis may occur, particularly as increased abdominal pain may cause a uterine discharge. The occurrence of fever would exclude dysmenorrhœa in cases in which this symptom was present. The pain and leg flexion simulate hip-joint disease.

After the first twenty-four hours, during which the above-mentioned symptoms described take place, the fever continues. There is anorexia, but vomiting occurs at longer intervals if at all. The local symptoms continue, although modified usually by methods of treatment which are applied. Both general and local symptoms frequently subside after a free movement of the bowels is secured, which occasionally takes place spontaneously. In other cases they continue a week or ten days, and may even extend over a longer interval of time.

After five or six days at the furthest, fever subsides, the local distention lessens, the paroxysms of pain disappear, and convalescence ensues. There may, however, be localized tenderness for a considerable period of time, and diarrhœa, or at least two or three evacuations each day, for a week or more. In rare instances peritonitis supervenes without the occurrence of perforation. The onset under these circumstances is more gradual, but the symptoms are like those of peritonitis under other circumstances. Infection takes place directly through the appendix.

When the fever continues, with mild diarrhœa, intestinal pain, and flatulency, the case may be mistaken for *typhoid fever*. The temperature is, however, more remittent in character in the former, and the diarrhœa is not characteristic of the latter. The eruption of typhoid does not occur, and the symptoms of the typhoid state do not ensue. The diazo reaction may aid in forming a conclusion. The occurrence of bronchitis and other symptoms of typhoid would point to the true disease.

Recurrent Appendicitis. Frequent attacks of mild appendicitis occur; they may occur as frequently as every three months, or the interval may be as long as a year. The attacks are similar to the attacks just described. The local symptoms in some instances are more marked, because there has been a localized peritonitis previously. The induration is greater, and dulness is more characteristic. In some instances the attacks are comparatively mild, continuing but twenty-four hours, and are described as attacks of colic. Often they have been treated by the patient himself, or by household remedies alone. The patient spends a night in agony with cramps, but the next day follows his usual habits. It is possible that there has been no fever with the attacks, but in all cases of appendicitis which I have seen fever has been a constant accompaniment.

Appendicitis with Perforation. Before perforation takes place the patient may have had symptoms of the mildest form of appendicitis for two or three days, or it may have extended over a long period of time, with the expression of colicky pains alone. Not being under observation, the presence of fever cannot be utilized as a diagnostic feature. The perforation may take place early in the course of an acute attack. After the characteristic symptoms of appendicitis just described, in their most intense degree, the symptoms of peritonitis set in. The abdomen rapidly becomes distended, the characteristic vomiting ensues, and collapse develops. Perforation under these circumstances has occurred within the first twenty-four hours, or at least has not been postponed beyond forty-eight hours. Local inflammation about the appendix does not take place, and the local signs of an inflammatory tumor are not present, although tenderness at the special point can be elicited. If the perforation is more gradual, and there has been time for the occurrence of local inflammation about the appendix, by which pus is prevented from infecting the peritoneum, or if perforation takes place behind in the connective tissue which surrounds the mass, in which situation there is always inflammation, the local signs of the abscess or inflammatory tumor occur. There is swelling of the affected side, the normal outline is effaced. The area is indurated, and although the early pronounced rigidity gradually gives way to a boggy sensation, œdema of the surface of the skin appears. This can be elicited by pressure on parts that are hard and resisting, as the spine of the ilium. Fluctuation can often be detected by bimanual palpation. Dulness is found, although in some instances it may be very slight, scarcely an appreciable change in pitch. Both light and deep percussion must be performed, and compared with the results of percussion in the opposite region. Examination per rectum may yield immediate results. An induration may be felt about the brim of the pelvis or the rectal fossa, which fluctuates and may eventually soften. With the finger in the rectum, and pressure above, better results may be obtained. If the symptoms of peritonitis do not arise, or rapid infection of the system take place, the signs of abscess become more and more marked. The surface becomes reddened, and pointing may take place toward the groin or opposite the spine. Sometimes the swelling increases in the direction of the loin, and the abscess may point in that situation.

As the abscess develops the general symptoms change. They now become the symptoms of *suppuration*. The fever is remitting or intermitting. There may be chills. Sweats are common, and there is loss of appetite and the occurrence of diarrhœa. In former times it was customary to see abscess develop in some other situation, or symptoms occur from burrowing of the pus in various directions. It may extend upward along the back of the colon, underneath the diaphragm, and from thence to the pleura and lung, and be expectorated. The abscess may open into the rectum or into the bladder. If the local inflammation is virulent and the symptoms are intense, if peritonitis has not taken place, the symptoms of septicæmia may rapidly ensue. This sometimes may occur quite early in the disease. There may be vomiting and septic diarrhœa, and a slight delirium at night. An excessively rapid

and feeble pulse is seen; in one instance seen it was irregular. Extreme prostration ensues, followed by the symptoms of the typhoid state.

It is clear that in cases of appendicitis we must attempt to recognize: (1) the inflammation before perforation has taken place; (2) the occurrence of perforation; (3) the occurrence of peritonitis due to either of the two conditions; (4) the occurrence of abscess (paratyphlitis and perityphlitis); and (5), the occurrence of septicæmia.

Typhlitis is an inflammation of the cæcum due to accumulation of fæces or foreign substances. It may be due to ulceration. The inflammation may remain as a localized enteritis, or may be followed by ulceration. In the majority of cases the ulceration is due to pressure by the contained foreign material or fæces. The inflammation occurs in early life usually. The patients have been subjected to constipation. The attack may follow some error in diet. There is pain in the right iliac fossa, constipation, and the occurrence of nausea. Moderate fever develops. On examination there is fulness in the right iliac region, and the right thigh may be flexed, the part is tender to pressure, and a doughy, sausage-shaped tumor may occupy the region of the cæcum. The more severe symptoms last two or three days. Local tenderness may continue a week or even longer. The tumor gradually disappears. If ulceration takes place, inflammation about the cæcum ensues. An abscess forms gradually in the flank behind. Perityphlitis is the term applied to this secondary abscess, although, as the term has been confused with paratyphlitis it had better not be used in this connection.

Appendicitis must be distinguished from perinephritic abscess and the abscess which follows perforation of the intestine or cæcum at this point. Perinephritis can scarcely be distinguished unless there has been a previous history of renal calculus and pronounced evidence of disease of that organ preceding the formation of the abscess. *Pericæcal abscess* follows the stercoral typhlitis which occurs as the result of cancer in the course of the large intestine. The history of their conditions point to the true nature of the disease. Abscess may occur behind the cæcum in cases of caries of the vertebræ and in some rare instances of empyema in which it has dissected downward. *Hip-joint* disease must be distinguished from appendicitis. The leg is flexed, the patient complains of pain about the region of the hip; unless careful observation has been made in the beginning of the attack the early march of appendicitis may not be recognized. The two are confounded after abscess formation. The flexed leg of appendicitis can be extended under ether, and examination then shows the joint to be free from disease.

Fenwick says that *acute tubercular peritonitis* may be confounded with perforation of the appendix. There are pain and tenderness in the hypogastrium, dulness on percussion, and fever. In tubercular peritonitis the onset is more gradual, the pain and tenderness more general, there is no distinct tumor or increased tension in the hypogastrium. If there is dulness on percussion, the line generally varies with the position of the patient. Diarrhœa is urgent, and there are, in most cases, some signs of consolidation of the lungs. The absence of tumor in the right iliac region and in front of the rectum is the chief point; for when

perforation occurs in phthisical subjects there is generally very slight pain, and severe diarrhœa is often the only prominent symptom.

Abscess about the head of the cæcum is due (1) to appendicitis, of which sufficient mention has been made; (2) to perforation of the cæcum on account of typhlitis; (3) to perforation on account of cancer of the intestine; (4) abscess secondary to kidney disease, perinephritic abscess; (5) to abscess secondary to disease of the vertebræ. The physical signs are those of abscess due to perforation of the appendix. The symptoms are the local symptoms of abscess and the general symptoms of suppuration.

Tuberculosis of the Intestine.

The disease is usually secondary to chronic tuberculosis, but may be primary, especially in children. The symptoms are usually those of diarrhœa, and in the primary form this is associated with general emaciation, which advances rapidly, and with anæmia. Fever of the intermittent or remittent type is present. There is meteorism; the abdomen is much distended, but eventually becomes contracted. The mesenteric glands can be made out along the spinal column, and the intestines may become bunched into a mass, yielding a dull tympany on percussion in the centre of the abdomen. The diarrhœa is attended with colicky pains. The diagnosis is based upon the rapid emaciation, irregular fever, enlargement of the mesenteric glands in a patient, usually a child, who had probably been exposed to tuberculous infection. In one of my cases the child, aged four years, ate of the same food, using the same utensils, as a brother, a lad of twenty-two, dying of pulmonary tuberculosis. The child was constantly with the brother. The remainder of the family, eight in number, remained in perfect health, and were all of good physique. The brother became infected by association with tuberculous subjects in improper quarters away from home.

Cancer of the Intestines.

The disease usually occurs late in life, and is associated with progressive *emaciation* and *cachexia*. There may not be any symptoms save general failure of health until the sudden occurrence of obstruction of the bowel. The symptoms vary with the position of the carcinoma and the direction of growth of the tumor. In some instances with the general symptoms there may be irregular *pain* in the abdomen, with irregularity of the stools. The *tumor* may be detected if the small intestine is involved. Its detection is facilitated by having the patient get on the hands and knees, palpating the abdomen in this position. If the tumor is seated in the lower colon, pain in the sacral region, resembling sciatica, may be complained of; if the cæcum or the sigmoid flexure is the seat of disease a tumor is usually detected. Wherever the situation, the tumor found is tender, usually lying in the axis of the intestine—movable if in the small intestine, fixed if in the cæcum or the sigmoid flexure. In the latter location the tumor may be felt per rectum. One notable characteristic is that it may be palpable some days and not be present at other times. The position and

size may vary from day to day, although it is always hard and knotty, not doughy. *Constipation* is characteristic of most of the cases. It may alternate with diarrhœa. The stools are frequently ribbon-shaped, or they may pass in scybalous masses, or large or small amounts of blood, chiefly the latter, are passed with pus or mucus; sometimes masses resembling cancer can be found in the stool. If the tumor is in the rectum there is great difficulty in defæcation; the act is attended by pain. Later the pain becomes constant, and may radiate to the hip or the genitalia. Sometimes this pain is the only symptom complained of. Mucus and blood appear in the stools, the bowels being alternately confined and loose. Paralysis of the sphincter ani may take place with incontinence. A tumor may be felt per rectum or be seen through the speculum. It may be a hard knotty mass.

The diagnostic symptoms are: (1) The general symptoms of cancer. (2) The tumor. (3) The occurrence of constipation which leads to complete obstruction, or obstipation, alternating with diarrhœa. Blood in the stools, with alteration in the shape of the fæces, is significant.

Amyloid Degeneration of the Intestines.

The symptoms are those of diarrhœa, persistent but mild in character, associated with symptoms of amyloid disease in other organs. With enlargement of the liver and spleen, changes in the urine due to amyloid disease are present. The occurrence of these symptoms in a patient with syphilis, or especially in a child with bone disease or tuberculosis, points to the nature of the case.

Infarction of the Bowel.

The symptoms take place suddenly. The patients have reached middle or late life, and have atheroma of the general arterial system. Sudden pain in the abdomen, with vomiting and symptoms of collapse, takes place. Moderate obstruction occurs with distention of the abdomen. After the pain diarrhœa sets in with the passage of blood. The age and the absence of tumor distinguish it from intussusception, the only intestinal condition with which it may be mistaken.

Dilatation of the Colon.

The dilatation takes place temporarily in constipation with obstruction. In rare cases it may become permanent. The distention of the abdomen is enormous. It may begin in childhood and continue through adult life. Congenital obstruction; the eating of oatmeal or similar food, with attendant constipation, leads to distention. The bowels are constipated. The constipation may continue for several weeks, during which period there is increasing dulness in the tract of the colon, with fæcal tumors distinguished by palpation. The constipation is relieved by diarrhœa, which may continue for two or three days, during which enormous amounts of fæces are passed. It may be preceded by vomiting of fæcal character. After the bowels are open the distention continues, the dulness being replaced by tympany.

Diseases of the Rectum.

Consideration of rectal lesions belongs to the surgeon. It is proper, however, to insist upon the very frequent deleterious effect of such lesions in neurasthenic subjects. Indeed, the bleeding which attends hemorrhoids may be sufficient to lead to profound anæmia, upon which neurasthenia may readily develop. The local suffering due to rectal fissure, or prolapse, may aggravate any tendency to the state of neurasthenia, or aid materially, with other conditions, to more firmly fasten it upon the system. In cases of anæmia, of neurasthenia, of the gastric neuroses, of debility, or prostration, the cause of which cannot be ascertained, the rectum should be examined. The appearances of hemorrhoids and other rectal affections are described in works on surgery. Hemorrhoids, ulcers, fistula, and carcinoma are to be sought for in abdominal affections.

Inspection and palpation are necessary. The symptoms are those of local pain, tenesmus, and frequently hemorrhage. The pain follows a movement of the bowels. There may be a feeling of a foreign body in the rectum, with some itching and burning about the anus. The pain may be so severe as to inhibit defæcation. The timid subjects will not endure the act; in consequence they suffer from vertigo, headache, tympanites and symptoms of gastro-intestinal disorder. In some instances there is chronic catarrh of the rectum with discharge of small stools containing mucus or pus streaked with blood. Cases occur in which hemorrhage is the only symptom, the constant recurrence of which leads to grave constitutional effects. Hemorrhoids are the lesions for which the rectum is most frequently examined. They, as well as other lesions, are of diagnostic significance in affections beyond the rectum. Thus, in all forms of portal congestion, internal hemorrhoids are of frequent and constant occurrence, and when found in the toper may be one of the first indications of cirrhosis of the liver. Rectal fissure is not of much diagnostic significance. The finding of a small cancer, the symptoms of which may be those of hemorrhoids, may explain emaciation and the development of cachexia. Ulcer of the rectum may be due to syphilis, cancer, or tuberculosis. A fistula is often tuberculous. The rectum must be examined in cases of pyæmia, particularly of the portal variety, when jaundice, enlargement of the liver, and hectic are present. Local rectal disease may cause pylophlebitis.

Diseases of the Peritoneum. Peritonitis.

Inflammation of the peritoneum may be acute or chronic. It may be general or localized. Acute inflammation is rarely primary; it may occur in the later stages of chronic Bright's disease, or other dyscrasia, without apparent cause. If it follows exposure to cold, or trauma, it is called traumatic peritonitis. It is due in the large majority of cases to extension from organs which the peritoneum covers, or to perforation of one of the abdominal organs. In the first instance it may follow inflammation of any portion of the gastro-intestinal tract, of the pelvic

viscera, and suppurative inflammation of the spleen and liver, and of the pancreas. In all instances the primary inflammation in the organs mentioned is due to some micro-organism, as the staphylococcus, the streptococcus, or the bacillus coli communis, and the peritoneal inflammation to extension of the infection. In a peritonitis that occurs from perforation, the element of infection also plays an important part, as in ulcer of the stomach or bowels. In inflammation of the gall-bladder perforation may take place with resulting peritonitis. Abscess in the liver, spleen, or kidneys, bursting into the peritoneum, also leads to general peritonitis. The most common forms, however, are due to appendicitis or disease of the Fallopian tubes. Acute peritonitis may also occur in cases of tuberculosis by direct infection.

Symptoms. The onset of acute peritonitis depends in a measure upon the cause. When there is perforation or infection the onset is sudden; chilly feelings or a rigor occur with intense pain in the abdomen. If at first localized the pain rapidly becomes general, is constant and increases in exacerbations, is very intense, aggravated by movements and by pressure. The patient lies on the back with the legs drawn up. The dorsal decubitus is assumed in order that the tension of the abdominal muscles may be relieved. The location of the pain depends upon the seat of primary infection; this is usually in the right or left lower quadrant, more marked about the tubes or the appendix. In perforation of an ulcer of the stomach the pain may be complained of in the back, or referred to the chest or the shoulders.

Physical Examination. On palpation the abdomen is extremely sensitive. The patient is unable to bear the weight of clothing or external applications. The abdomen gradually becomes distended, and on percussion is tympanitic. The distention may become so great as to push up the diaphragm and interfere with the respirations, so that they are shallow; and dislocate the heart so that the apex beat is seen in the fourth interspace. The splenic dulness may be obliterated entirely and the liver dulness reduced. It is said that in some instances this may be obliterated, although recent observations affirm that such obliteration only occurs in the anterior portion of the abdomen. Liver dulness persists in the axillary region, though diminished in extent. This obliteration could only take place in perforative peritonitis. Osler points out that in pneumo-peritoneum, perforation may obliterate the hepatic dulness, although dulness in the lateral region continues on account of the effusion of fluid. If a patient with gas in the peritoneum is turned on the left side a clear note is heard beneath the seventh and eighth ribs (hepatic region). The abdominal muscles are often rigidly contracted. In some cases, usually when the inflammation is due to the streptococcus, there is not much distention of the abdomen, or it may be flattened entirely with board-like rigidity. In these instances pain is not so marked, and tenderness may not be complained of.

The *respirations* are hurried and the superior thoracic type of breathing is seen because the action of the diaphragm is painful. The act of speaking or coughing increases the pain, and the patients are unable to take a full breath without suffering. With the occurrence of pain and local signs, *vomiting* usually sets in. It is painful and at first is com-

plete, the contents of the stomach being ejected and then a yellowish bile-stained fluid; later the vomit becomes greenish in color. Complete vomiting is displaced by simple regurgitation of fluid, so that on the slightest motion of the patient, or on taking a small amount of fluid, the characteristic greenish-colored fluid is regurgitated without action of the diaphragm. This may for twenty-four to forty-eight hours be almost continuous. The *tongue* is moist and furred early, but later becomes dry and often is cracked and red. The *bowels* are constipated. They may be loose at first, but constipation is characteristic. The intestines are paralyzed from overdistention and from œdema of the walls due to inflammation.

The general symptoms are marked. After the chill the temperature rises to 104° or 105°. In septic cases it continues at this point, or may rise to a greater height. If cases progress rapidly a temperature of 105° or 106° on the second or third day is not uncommon. In other cases after the initial rise the elevation subsequently is not so great, but there is not much difference between morning and evening temperature unless there is an abscess.

The *urine* is scanty; micturition may be frequent and painful, particularly if the inflammation began in the pelvic organs. The urine usually contains a large amount of indican in the suppurative form.

The appearance of the patient at the height of the disease is characteristic. The expression is anxious, the face is pinched, the eyes sunken. Vomiting causes wasting. The collapse is marked, with the characteristic facies previously described (see Expression). The pulse is rapid and feeble and soon becomes thready, ranging from 110 to 150. In the first stages it may be small and hard. Attention has been called frequently to the peculiar wiry pulse of the early stage of peritonitis.

In severe cases death may take place in thirty-six to forty-eight hours. Usually a fatal termination does not take place for five or six days, and it may be longer. The vomiting persists, collapse with falling temperature ensues, the pulse becomes rapid and thready. Throughout the entire attack, unless symptoms of septicæmia are marked, the mind is clear. The patient dies of paralysis of the heart. Septicæmic symptoms are indicated by a dusky color of the face, rapid and irregular pulse, slight delirium, dry brown tongue and other evidences of the typhoid state.

If the cases are prolonged some effusion may take place into the peritoneal cavity. Dulness is noted in the flank, and if it is possible to move the patient it alters the position. If recovery takes place, particularly in tuberculous cases, the affection may become circumscribed and be indicated by dulness which is not movable.

Diagnosis. It is essential in making a diagnosis to ascertain, if possible, the primary source of the infection or inflammation. Inquiry in order to determine this is made with regard to the age, sex and history of previous disease of the patient. In young adults appendicitis is first to be thought of; in females inflammation of the pelvic organs. In chlorotic subjects, if the pain is high up, the history of ulcer of the stomach must be inquired for. Later in life, particularly if there has been jaundice, the history of frequent attacks of gall-stones and of

hepatic disturbances must be ascertained. All forms of intestinal obstruction must be sought for. Frequently, however, a definite cause cannot be ascertained. If it occurs in the course of typhoid fever it is usually due to perforation, but the occurrence of pain may not be complained of on account of the mental state of the patient. Under other circumstances the symptoms cannot be overlooked.

Acute peritonitis must be distinguished from entero-colitis. The distinction is not usually difficult to recognize if attention is paid to the development of the case. The pain is not so severe in entero-colitis; it is more colicky in character. The general tenderness is not so great as in peritonitis, and the distention does not interfere with respiration to such a marked degree. Diarrhœa is more common in entero-colitis; collapse is not so pronounced if present. *Obstruction of the bowel.* The diagnosis is difficult in the absence of a distinct history, but in peritonitis we do not have stercoraceous vomiting. The tympanites is more general, the pain is more general, and the vomiting is different unless the peritonitis is due to obstruction. A tumor, if present, may point to the true nature of the case, and if there is any discharge from the rectum, invagination may be the exciting cause.

Peritonitis is simulated by a condition to which the name *hysterical peritonitis* has been applied. It occurs in hysterical subjects, and in every feature the true form is imitated. The mode of onset, the decubitus, the difficulty in micturition, and the local distention and tenderness of the abdomen are characteristic of both. In a few cases which we have seen the vomiting is not of the nature of true peritonitis, either in the mode of ejection or the character of the fluid. It must not be forgotten that even the temperature may be elevated and collapse take place in the hysterical form. In the cases which I have seen the abdominal facies does not develop, while, on the other hand, the facies of hysteria, with the self-interest which the patient exhibits and the precision with which symptoms are narrated, coupled with emotional or other manifestations of hysteria, point to the true nature of the affection. Other symptoms of hysteria may arise. The case is judged by the history of these associated manfestations and the permanent stigmata of the disease. There is always a positive absence of cause, and of disease in any of the abdominal viscera. Sometimes in these cases if the attention of the patient is diverted the tenderness on pressure may not be complained of. I am not familiar with the results in examination of the urine in this form of peritonitis. Indican should not be increased necessarily, as we find it in acute suppurative peritonitis.

Rheumatism of the Abdominal Walls. There is absence of a history of sudden acute pain followed by general pain. The fever is not so great. The respirations are not interfered with, the pulse is not so rapid, and symptoms of collapse do not supervene. A rheumatic pharyngitis, or inflammation of muscles in some other portion of the body may occur simultaneously. *Acute hemorrhagic pancreatitis* may simulate peritonitis in the sudden intensity of pain and the occurrence of shock.

Local Circumscribed Peritonitis. The causes of localized peritonitis are those of general peritonitis—that is, extension of inflammation from neighboring viscera, or perforation of the viscera. In the latter

instance the inflammation does not become general, because of rapid local inflammation shutting off the perforated area from the general cavity of the peritoneum. Local peritonitis of mild degree and local or circumscribed peritonitis with suppuration, are therefore found in the neighborhood previously indicated, from which a general peritonitis may start. The inflammation, however, if retained by a limiting wall may, after suppuration has taken place, gradually extend and the pus burrow in various directions. In such cases of localized peritonitis as may exist in the upper half of the abdomen, a sub-diaphragmatic abscess may form, or an abscess containing air and pus, known as pyo-pneumothorax subphrenicus. If the inflammation is secondary to disease of the pancreas it may be limited to the lesser peritoneum and cause the physical signs of effusion in this cavity. (See Disease of the Pancreas). Sub-diaphragmatic abscess is not limited to the lesser peritoneum. It can only be recognized by the history of previous disease on account of which perforation may take place, and by the general symptoms of abscess. If the abscess is on the left side there is extension of dulness upward toward the scapula, the lower limit of the lungs in health ceasing at the eighth or ninth interspace. There may also be dulness in the axillary region. If the abscess is on the right side it may simulate enlargement of the liver and be characterized by marked increase in dulness anteriorly, laterally, or posteriorly. Localized peritonitis in the lower half of the abdomen is due to disease of the vermiform appendix, or to disease of the Fallopian tubes. The localized signs are, first, those of pain and tenderness; second, the development of tumor.

Chronic Peritonitis. The symptoms of diffuse peritonitis, chronic in course, may follow the acute, or may occur in the course of tuberculosis. The intestines and peritoneum are matted together. General pain and tenderness, with a prolonged period of ill health, attend the diffuse form. (See Tuberculous Peritonitis.) In the chronic forms, particularly if there is considerable fibrous proliferation independent of cancer and tubercle, the abdomen becomes retracted, the muscles rigid, the note over the abdomen modified or dull tympanitic. The modification may be detected, in the upper half of the abdomen particularly, and especially over the liver. Sometimes a fremitus can be felt. The patients are under weight and without strength. The pain may continue a long time. It finally results, at least clinically, in such compensation that the patient is able to continue his usual occupation. Localized bands form, and may cause local sensations of a dragging character, or pain with drawing or pulling sensations, but, save the local symptoms, these are not serious, unless it should happen, as has been seen in intestinal obstruction, that coils of intestine are twisted about the bands or caught in them, leading to obstruction.

Ascites.

Ascites is the accumulation of fluid in the peritoneal cavity. The causes may be local or general. It occurs, first, in simple, cancerous or tuberculous inflammation of the peritoneum; second, in portal obstruction from disease of the liver, as cirrhosis, or disease of the portal

veins, either from compression or inflammation. Tumors of the abdomen are often attended by ascites, particularly solid tumors of the ovary. The *general* causes of ascites are the causes of dropsy.

Symptoms. The abdomen is enlarged, the enlargement being uniform. The skin is tense if the effusion is large, and *lineæ albicantes* may be seen. The navel may project. If the ascites is due to liver disease or disease of the portal vein the superficial veins may enlarge, although the enlargement is sometimes seen when any effusion continues a long period of time. On palpation fluctuation can usually be detected. Care must be taken not to confound the wave of the abdominal walls, produced by percussion, with the wave of the fluid underneath; the former must be cut off by the hand of an assistant placed vertically in the median line. The left hand should be applied firmly against one side of the abdomen, while with the right percussion or tapping is gently performed at the opposite point. The points selected should be at about the level of the fluid. At first the hand should be placed on the flank, and if the fluctuation is not revealed, then with each successive percussion it should be brought forward toward the median line. Sometimes light percussion will yield the sign, at others more firm percussion must be employed. In order to ascertain the position of solid organs in ascites, *dipping* is employed by suddenly pressing the tips of the fingers over the organ sought for. The fluid is thus displaced and the edge or surface of the organ readily felt. The faintest tap may be sufficient.

Percussion. When the abdomen is *percussed* in the usual manner there is dulness over the fluid. As the fluid gravitates to dependent portions the dulness is found in these portions. When the patient is lying down it is in the flanks, and may extend around the lower portion of the abdomen. If the patient stands up the dulness may reach to the umbilicus in the median line and to the same level in the mid-clavicular line. The *subjective symptoms* are those due to the cause of the ascites and to mechanical pressure. In ascites it is important to ascertain the nature of the fluid. This can only be done by aspiration. If the fluid is serous it has the characteristics belonging to that fluid. Hemorrhagic effusions usually occur in cancer and tuberculosis, although both of these diseases may occur with clear serum. In ruptured tubal pregnancy the effusion is hemorrhagic. In rare cases a chylous, milky fluid is found in disease of the lymphatics. In one instance this occurred from perforation of the thoracic duct. Chylous ascites may, however, be due to an excessive milk diet. In other instances it is due to filaria. The patient on a milk diet is often lipæmic, in consequence of which effusions are made turbid.

Ascites must be distinguished from enlargement of the abdomen due to ovarian tumor, enlargement due to pregnancy, and enlargement due to an overdistended bladder. In ovarian tumor the development at first takes place to the right or left of the median line. If enlarged the signs of it may be in the central region of the abdomen. The flanks, however, are always tympanitic on percussion. On examination *per vaginam* the local disease may be ascertained. A distended bladder should always be thought of, and catheterization performed in cases of

doubt. Cysts of the pancreas may be mistaken for ascites, and large hydatid cysts connected with the liver may simulate an accumulation of fluid in the peritoneal cavity. The history and the appearance of the fluid on aspiration point to the diagnosis.

Cancer of the Peritoneum.

It usually occurs in the aged, and follows cancer in other organs, as the stomach, liver, or uterus. Occasionally it is primary. The omentum is indurated and forms a mass which lies transversely across the abdomen in the upper zone. Ascites usually develops, and the exudation is bloody. The disease occurs more frequently in women than in men. With the development of ascites there is emaciation. The surface of the indurated omentum is irregular. It may be painful on pressure. The same character of tumor is seen in tuberculous peritonitis, and I have seen several such tumors in the aged without apparent cause, unless from proliferative peritonitis. (See Tumor.) Progressive emaciation, chronic ascites without cause, and a localized tumor without the occurrence of fever, point to the probable nature of the case. Sometimes pain is the most pronounced symptom. If these symptoms are present without symptoms of disease in other organs, as the stomach, rectum, or uterus, there is probably cancer of the peritoneum.

Tuberculosis of the Peritoneum.

The tuberculous process in the peritoneum may be either acute or chronic. In some instances it may continue without any symptoms, either local or general. Acute tuberculous peritonitis may exactly simulate suppurative peritonitis, save that the course is more prolonged and the fluctuations of temperature less pronounced. In other respects it cannot be distinguished from acute general peritonitis, save in the absence of the causes of the latter. A history of liability to tuberculous infection, or the presence of tuberculosis in some other portion of the body, may be of service in determining the nature of the case. This is sometimes impossible. Usually there occurs in a short time associate tuberculosis of other serous membranes, so that tuberculous pleurisy or tuberculous pericarditis will supervene, an associate process which does not take place in ordinary peritonitis. At the same time in most cases there is a diarrhœa—at least this has been present in the few instances in which I have seen this form of tuberculosis.

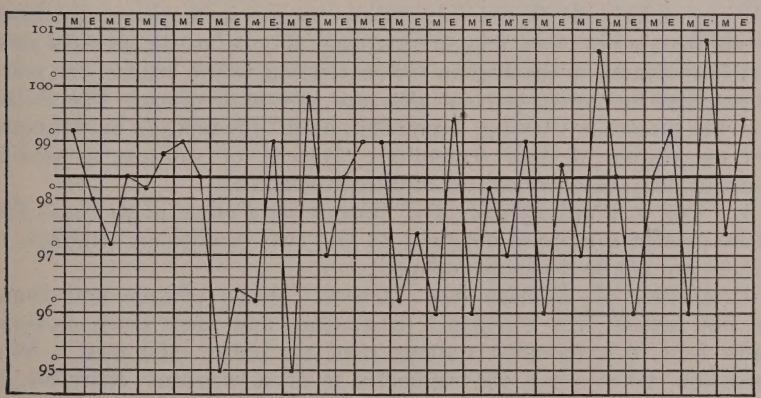
Acute tuberculosis of the peritoneum may precisely simulate acute appendicitis in, first, the local symptoms and signs; and second, the subsequent infection of the peritoneum. In acute tuberculous appendicitis, however, the signs of a tumor are not so marked as in true appendicitis. Nevertheless, in one instance, Keen operated upon a patient of mine, a healthy laborer in a rolling-mill, who had the classical symptoms of appendicitis. At the operation the appendix was found to be perforated and hanging in a local abscess. A fæcal fistula

ensued which did not heal, and within two months the patient died of general tuberculosis. The appendix was the seat of primary tuberculous ulceration.

In the second instance the appendicitis arose in the course of tuberculosis.

In the third instance, the patient, aged forty-five, was admitted to my wards in the Philadelphia Hospital, with high fever and pain in the abdomen, at first more pronounced along the margin of the liver. It became more decided by the end of twenty-four hours in the right lower quadrant of the abdomen; tenderness at McBurney's point was distinct, the area was enlarged, dull on percussion, the surface slightly œdematous. Fluctuation could not be detected. Extension of the leg was painful. Rapid general peritonitis ensued, during which the surgeon saw him, but declined to operate until the subsidence of the attack. When the attack subsided the local signs of tumor were not present. The fever persisted irregularly for a short time, indeed the more acute peritoneal symptoms subsided; then the right pleura became infected, and cough ensued with expectoration of muco-purulent fluid. It did not contain bacilli, however. Subsequently, the left pleura and the pericardium became involved. During the entire course of the disease there were diarrhœa, most pronounced sweats, rapid emaciation, and exhaustion. At the end of five weeks death took place, and at the autopsy general serous tuberculosis was found to be present.

FIG. 88.



Tuberculous peritonitis. Subnormal temperature.

While in a number of instances the symptoms are acute and alarming, in the larger proportion of cases the process is more chronic, and is attended by characteristic local and general symptoms. In the prolonged and moderate cases there may be continued fever of moderate degree, or it may be remitting in type. In old people the fever is frequently subnormal (see Fig. 88). With the fever there is more or less rapid emaciation. The sweating is profuse and characteristic. In more severe cases the temperature is high but irregular in type,

approaching more the remittent form. The general symptoms very much resemble typhoid fever. Indeed, symptoms of the typhoid state may ensue.

The Local Symptoms. Four classes are seen: (1) Abdominal enlargement with effusion; (2) enlargement with tumors; (3) combination of the two; (4) enlargement without marked evidence of fluid or tumor in the abdomen. In this form and in the forms in which tumors are present, the abdomen subsequently may undergo retraction.

1. *Enlargement with Effusion.* The local symptoms and physical signs are those of ascites. The abdomen is never so distended, however, as in the ascites of cirrhosis of the liver. Often the fluid is not moveable on account of adhesions which may be distinctly localized in the right or left quadrant of the abdomen, in which situations fulness and fluctuation may be readily detected.

2. *Tuberculosis with Tumors.* The tumors are usually in the upper zone of the abdomen, and may be localized to either quadrant, or extend from the right to the left. They are usually due to tuberculosis of the omentum, with secondary contraction. In some instances a hard, indurated tumor, somewhat tender on pressure, may extend across the abdomen midway between the xiphoid cartilage and the umbilicus. It may be as low as the umbilicus, and vary from two to four inches in width. It may be continuous with the liver dulness. In other instances more distinctly localized masses may be felt. These may be to the right or to the left of the umbilicus. In other instances they are hard, slightly tender, with an irregular surface. They may be movable and alternate with the change of position of the patient. I have never seen tuberculous masses in the lower quadrants. In children with tabes mesenterica they may be made out close to the vertebral column in the median line, extending to the brim of the pelvis, although at the lower portion they are not so distinct. The dulness over the tumors is varying, dependent upon the relation to the bowels and the degree of their distention. Instead of dulness, a modified tympany may be observed, or muffled resonance.

3. *Cases in which Effusion and Tumors are Present at the Same Time.* These present symptoms common to the two conditions, although the tumors are not so distinctly defined.

4. *Absence of Effusion and Tumors.* When effusion and tumors are not present, the thickened peritoneum and more dense intestinal walls lead to a modified dulness over the entire abdomen. When retraction takes place the resonance is of a woodeny character, the abdomen is more or less tender, and ill-defined indurations may be present. The term *carreau* is applied to these indurations. In not a few instances the local physical signs may apparently be due to inflammation of the liver on account of extensive perihepatitis. In one case of a child, the local signs during life were of this character, and the symptoms were simply those of loss of appetite, with discomfort and weight and fulness below the sternum. Both the right and left lobes of the liver were covered with an enormous thickening due to tuberculous inflammation. Simple plastic peritonitis occupied the lower zone. Apart from the general symptoms and the local physical signs the other symptoms are not distinct save those due to tuberculosis in other

situations. The appetite is usually poor, there is some atonic dyspepsia, vomiting may occur at regular intervals; the bowels may be constipated, although in my experience they have usually been relaxed. The patient becomes anæmic, the skin harsh and dry. Emaciation may progress to an extreme degree Eruptions and boils may break out, some œdema of the ankles may occur. Death takes place from exhaustion, and from the development of tuberculosis in other localities.

The *diagnosis* is difficult. The two extremes probably present the greatest difficulties. The age also modifies the ability to make a diagnosis. Peritoneal tumors with or without effusion in young subjects are almost always due to tuberculosis. In the aged they must be distinguished from carcinoma or chronic peritonitis from other causes. The association of diarrhœa with the symptoms is rather against carcinoma. Sacculated effusions may be confounded with abdominal tumors, as of the ovary. The resemblance is more pronounced if the tubercles develop primarily in the tubes or uterus. In a recent case the autopsy disclosed a large caseating ulcer inside of the uterus, and tuberculosis of the Fallopian tubes and peritoneum. The right tube was chiefly affected. The effusion during life was sacculated in the right quadrant, was not movable with the patient, and fluctuated both on external palpation and with bimanual palpation *per vaginam*. It was impossible to distinguish it except that there was dulness instead of resonance in the flanks. As Osler has pointed out, the association with salpingitis must arouse suspicion, particularly if at the same time disease may be found in some other organ of the body, as the apex of the lung or the pleura. In males, the primary lesion is often in the testicles. The history of the case and the development of the disease in an irregular manner, associated with gastro-intestinal disturbance rather than disturbance of uterine function, are points in favor of tuberculosis. Tympanites is of frequent occurrence.

CHAPTER VI.

DISEASES OF THE LIVER, SPLEEN, AND PANCREAS.

THE symptoms of disease of the liver are due to the morbid process which affects the organ, to disturbance of the functions of the hepatic cells, or to obstruction of the channels for the flow of blood and bile. As the latter are beyond the glandular structure of the liver they may be affected by disease outside of the liver. Hepatic symptoms may, therefore, be due to disease outside of the liver.

The morbid process may, in time, cause alteration in function, obstruction of channels, or physical alterations in the size and shape of the liver. The latter may also occur from disease outside of the liver.

SYMPTOMS DUE TO THE MORBID PROCESS. The *morbid processes*, on account of which symptoms are created, are chiefly congestion of the liver, abscess, cancer of the liver, and the degenerations.

In *congestions* of the liver the symptoms are (1) the symptoms of the cause, (2) enlargement of the organ on account of increased amount of blood, (3) functional disturbance for the same reason. The congestion is not limited to the vessels in relation to the liver cells, but involves the vessels of the mucous membranes also, hence the latter are swollen, by which the ducts are obstructed and jaundice is produced in moderate degree. In *abscess* of the liver we have the symptoms of suppuration and changes in the shape of the organ. Modifications of its function are not observed, and obstruction of the channels rarely takes place. In *cancer* of the liver the symptoms are those of malignant disease in general, to which are added symptoms due to change in the size of the liver, and, more frequently than in abscess, symptoms due to obstruction of the channels. The *degenerations* are so frequently secondary to and masked by the symptoms of their primary cause that, save in regard to change of size, there are no hepatic symptoms of note.

SYMPTOMS DUE TO FUNCTIONAL DISTURBANCE OF THE LIVER. The functions of the liver are, first, to secrete bile; second, to destroy the hæmoglobin of the blood; third, to destroy poisons entering the portal circulation through the intestinal tract, or modify their character. Bile is not secreted when the liver cells are destroyed, as in acute yellow atrophy; giving rise to jaundice, hemorrhages, and grave cerebral symptoms. In this case the liver does not destroy the normal amount of hæmoglobin. On the other hand, hæmoglobin may be so much in excess that the liver cannot destroy it; hence jaundice is created (see Hæmatogenous Jaundice). Functional disturbances of the liver are seen clinically when products of digestion are not completely destroyed by the liver and are permitted to enter the circulation. In this manner we have, on the one hand, possibly, the occurrence of glycosuria; and, on the other, the occurrence of lithæmia or other toxic states.

Lithæmia is the more common condition believed to be due to liver disturbance. There is an excess of uric acid and urates or of other metabolic compounds in the blood. The *symptoms* that are produced are, first, symptoms of excess of lithic acid in the system; second, the effects of the lithic acid upon the nervous system. Lithæmia may be acute or chronic.

Acute Lithæmia; Biliousness. When acute, the local disturbances are those of furred tongue, a bitter taste in the mouth, anorexia, nausea, disgust at the sight of food, with possible morning vomiting. There is some tenderness in the upper mid-abdomen, and after eating, weight and fulness and distress in that region. Flatulency occurs. The symptoms of intestinal dyspepsia may arise secondarily. Slight fever or feverishness may attend the attack. The skin is hot and burning; or cold perspirations may break out at irregular times, alternating with flashes of heat. The bowels are constipated, the stools are clay-colored. The symptoms may be attended by slight obstruction to the ducts on account of which jaundice prevails in moderate degree. In some instances the liver can be made out slightly enlarged. The urine is loaded with urates and uric acid. It is scanty and high-colored, and there may be painful micturition. The *nervous symptoms* are usually those of depression, as headache, some dulness, or stupor; the patient may be unusually drowsy. The headaches may be the most prominent feature of the attack. They are frontal, attended by slight vertigo, flashes of light or spots before the eyes, and ringing in the ears.

The same group of symptoms is seen in acute gastro-duodenal catarrh.

Chronic Lithæmia. In *chronic lithæmia* the symptoms are variable and are characterized by disturbance of function in nearly all of the organs of the body. They have been classically described by Murchison, Da Costa, and others, and while the theory is fairly satisfactory to work upon for lines of treatment, the same group of symptoms may be met with in forms of chronic indigestion, particularly the forms in which there is inability to digest sugars and starches. By some the symptoms are attributed to chronic intestinal catarrh.

Symptoms. The patients are in ill health and subject to chronic indigestion. They may be under weight or corpulent. The skin is harsh and dry, its nutrition poor. It is subject to erythema. The condition of the skin is such that local inflammations, as eczema, may arise. Irregular sweats occur, alternating with periods of hot, dry skin. The extremities are cold and clammy, and tingling and numbness are often complained of.

Gastro-intestinal Symptoms. The symptoms are those of chronic indigestion There is constantly a furred tongue with local dyspeptic symptoms. The bowels are irregular or constipated; sometimes mucus is passed. Flatulency is prominent and marked, both gastric and intestinal. A slight icteric tinge may be seen on account of a slight local catarrh of the ducts or hepatic congestion. It recurs at frequent periods, while a sallow complexion is more or less constant.

Respiratory Symptoms. The patient is liable to attacks of catarrh of the upper air-passages, and especially to pharyngitis. In lithæmic

states tonsillitis is not uncommon. Chronic pharyngitis is present. On the other hand, some persons, particularly those over fifty, have chronic bronchitis, and attacks of asthma are common. It cannot be distinguished from bronchitis due to other causes, except by the fact that the subject is lithæmic. Emphysema of the lungs develops on account of bronchitis and tissue degeneration.

Cardiac Symptoms. Palpitation of the heart is a constant accompaniment of lithæmia in many states; in others there may be unduly rapid action of the heart, or, during exacerbations, slowness of the heart's action. In the later stages, pseudo angina pectoris is of common occurrence. In the earlier stages pain about the heart or in the left side is frequently complained of.

Nervous Symptoms. Constant headache, worse in the mornings, relieved in the after part of the day. Some vertigo may be present. There is depression of spirits and inaptitude for mental exertion. The memory is dull, the faculties blunted. The patient is subject to backache; the pain is chiefly marked in the loins. Pain in the right shoulder is of frequent occurrence. In addition, pains along the course of the nerves (neuritis), and myalgias, are of common occurrence. The nerve-trunks may be tender. There is tenderness in the sheaths of the muscles, or at the insertions of fascia and tendons. Peripheral nerve-sensations are common. Numbness and tingling are frequently complained of. Paræsthesiæ of all forms, variously distributed, are a source of annoyance. Local sensations of heat or burning alternate with areas of coldness. Tingling, pricking of needles, and other forms of paræsthesia occur.

The Urine. In this class of cases the urine is high-colored and contains an abundance of uric acid and urates. The amount is scanty, the specific gravity high. There may be albumin, small in amount, depending upon the irritation of the urates in their passage through the kidneys. Cylindroids are present in the urine; casts are not common, although at times when the uric acid is passed in excess there may be a secondary nephritis, with albumin, blood, and casts. As the ultimate results of such condition we find the development of gall-stones, or of calculi in the kidneys and bladder. Lithæmic patients are subject to attacks of hepatic or renal colic.

A further ultimate result is gout or rheumatism. Acute inflammatory rheumatism (rheumatic fever) does not belong to this category, but muscular rheumatism, subacute inflammation of the joints with moderate fever, true gout, and gout with its modifications when seated in the various joints, are the ultimate outcome of this process in the patient. Attacks of gout may occur in a patient who has not presented symptoms of lithæmia, but those who have symptoms of lithæmia are more susceptible to causes which produce attacks of gout. The gouty and rheumatic manifestations are due to the deposition of uric acid and urates in tissues which are not highly vitalized, and in which, therefore, the circulation is sluggish.

Lithæmic states later assume the *gouty aspect.* Tophi are seen in the situations natural to them. The appearance of the face is characteristic, with capillary congestions and stases. The patients usually become

more or less obese and are subject to attacks of glycosuria. Early in their life degenerations of vessels take place. The kidneys are always under an excessive strain. A considerable portion of material is not discharged that should be; its effects upon peripheral vessels are such as to cause heightened tension, therefore undue vasomotor congestion of the vessels takes place, leading to low-grade inflammations, with the development of atheroma. For the same reason, chronic interstitial nephritis is set up, and because of heightened strain in the vascular system, chronic sclerotic valvulitis.

Functional symptoms from disorder of the liver are otherwise not marked, unless we include a group of cases in which sudden coma and convulsions take place, presumably because material has been absorbed from the gastro-intestinal tract and enters the general circulation because of the abeyance of the function of the liver, the office of which is to destroy the material. Such symptoms may arise in organic disease of the liver, as cirrhosis.

Symptoms due to Obstruction of the Channels. The symptoms are produced by disease of the channels or by disease outside of the channels, as in obstruction of the bile-ducts by pressure. (1) Obstruction of the *bile-ducts* causes jaundice, at times pain, and at times fever. The three symptoms may occur singly or combined. Jaundice may occur alone in obstruction by *gall-stones;* pain may occur with it, or jaundice, pain, and fever may occur together; rarely, pain or fever may be present alone. Each symptom will be described later. Obstruction of the *blood channels* causes hyperæmias of the liver and portal congestions. The symptoms of each will be discussed; suffice it to say that here again the symptoms are modified by the process. Thus, in portal obstruction from pressure, the symptoms are far different from the symptoms of portal obstruction due to suppurative inflammation of the vein.

(2) Obstruction to the flow of blood takes place in hyperæmia, which may be either active or passive, and in disease of the portal vein, rarely of the hepatic.

Hyperæmia of the Liver. In the *hyperæmias* the liver is enlarged. If the hyperæmia is active, painful distention may be complained of, and the organ may be the seat of some tenderness. There may be, in addition, weight and fulness in the liver region. Active hyperæmia may follow a chill or suppression of the menses, but more frequently occurs after indiscretions of diet, the free use of alcohol, or stimulating food followed by an attack of acute gastro-intestinal catarrh. It is more common in the tropics, and is due in that climate to suppression of the perspiration. It is recognized by the occurrence of symptoms of acute gastritis with enlargement, pain and tenderness of the liver. Slight jaundice may attend the attack. *Passive congestion* is also attended by enlargement of the liver. The enlargement may cause a sense of weight or fulness, but pain is not complained of. The organ is not tender, the edges are smooth and indurated. The liver may pulsate. This is detected when the hand is placed over the surface of the liver, when, with each impulse of the heart, the organ can be felt to expand. The symptoms of the cause of the passive congestion combine

with those just narrated due to enlargement of the organ. To them must be added symptoms due to obstruction of the flow of blood in the portal circuit. Passive congestion occurs in organic heart disease after compensation has failed and the right heart is dilated. The organ rapidly becomes congested because of its close proximity to this chamber. In emphysema of the lungs, in fibroid phthisis, in intra-thoracic tumors pressing upon the vena cava, mechanical congestion takes place. The recognition of passive congestion is not difficult. The symptoms due to enlargement (see Objective Symptoms) and the symptoms due to portal obstruction point to the true nature of the hepatic lesion.

The Symptoms of Portal Obstruction. These arise because of disease of the portal vein, or because of occlusion and obstruction to the flow of blood in the branches of the veins. The diseases of the portal vein are *thrombosis*, *adhesive* and *suppurative inflammation.* (1) *Thrombosis* of the portal vein attends cirrhosis of the liver, and may occur secondarily to pressure upon the vein by a tumor within the abdomen. Disease of the pancreas was the cause of the pressure in a patient under my observation. As a result of the thrombosis, adhesive inflammation of the vein takes place, to replace which a collateral circulation is established.

The symptoms of *disease of the trunk* of the portal vein which leads to obstruction of this character are the same as in obstruction of the terminal branches, and are known as the symptoms of portal congestion (see below). In one respect only do they differ. While in both we have ascites, in thrombosis of the portal vein it occurs suddenly, and is characterized by rapid recurrence after tapping.

(2) *Suppurative inflammation* of the portal vein is attended by symptoms resembling pyæmia; the condition is called portal pyæmia. The inflammation is secondary and depends upon inflammation in the portal area. It follows appendicitis with peritonitis, ulceration of the hemorrhoidal veins, inflammation of the veins from ulceration or suppuration anywhere in the gastro-intestinal tract. The enlarged portal vein being the seat of suppuration it naturally follows that pus is carried into the liver. In consequence thereof, multiple hepatic abscesses arise. Three pathological affections are therefore seen: (1) Suppuration in the portal area; (2) during the height of the latter, or subsequently, symptoms of pyæmia develop, chills and fever and sweats, followed by exhaustion; (3) the occurrence of multiple abscesses of the liver (for the symptoms of which see Abscess).

Symptoms of obstruction, due to congestion, overfilling, or obstruction of the branches in the liver. This condition occurs in passive congestion, but most typically in cirrhosis of the liver. The circulation of the liver being interferred with, the blood is thrown back into the portal vein and the other end of the portal circuit. As a result we have (1) congestion of the mucous membrane of the stomach and bowels, with the symptoms of gastro-intestinal catarrh; (2) dilatation of the veins, chiefly the hemorrhoidal, on account of which hemorrhoids develop; (3) the occurrence of ascites; (4) the occurrence of hemorrhages. Hemorrhages due to disease of the liver may occur in any portion of the gastro-intestinal tract. Hæmatemesis and intestinal hemorrhage occur. The vomiting of blood may be in small amounts, associated only

with the discharge of mucus. In some cases large hemorrhages take place either from the mucous membrane of the stomach or from the veins about the œsophagus, which often become varicosed in cirrhosis. Hemorrhages from the intestine may occur from enlarged hemorrhoidal veins, from an intestinal ulcer which may be present, or from the mucous membrane of the intestinal tract. (5) Enlargement of the spleen. (6) The changes due to the establishment of the collateral circulation. If complete collateral circulation is established the above symptoms may not ensue. The *collateral circulation* may occur in deeply seated veins, or be established through the veins over the surface. If the latter, the external veins of the abdomen are enlarged. The epigastric and mammary veins become prominent. At times the veins about the umbilicus distend, and they may become so enlarged and prominent as to form a swelling to which the term *caput Medusæ* has been applied. The venules along the line of attachment of the diaphragm in the lower thoracic zone are overdistended.

On account of the enlargement of the terminal branches of the portal vein in the liver they press upon contiguous structures and interfere with the circulation of blood in the major vascular system of the liver, and hence invite a catarrh of the terminal ducts, on account of which they are obstructed, and slight jaundice supervenes. This is seen quite frequently in passive congestion of the liver, rarely in cirrhosis.

Symptoms due to Changes in Shape and Size. The liver may be enlarged, contracted, or irregular. (See Objective Symptoms.) Symptoms of portal obstruction occur when the liver is contracted.

The Data Obtained by Inquiry.

A number of extraneous factors are of aid in the diagnosis of hepatic affections. In disease of the liver more than in that of any other organ of the body we find the affection secondary to disease elsewhere. Moreover, diseases of the liver are almost always associated with pronounced and definite causes, the presence or absence of which are of great diagnostic significance. In the study of hepatic disease we consider, therefore, among etiological factors, the age of the patient, the sex, the habits of life, the climate, and the presence or absence of disease in other portions of the body. Primary liver disease is comparatively rare. Secondary liver disease, on the other hand, is of common occurrence. But few general diseases or states of the system occur that do not in some way influence the liver. The above remarks refer to organic disease. Separation of functional disorders, as previously remarked, from functional disorders of the stomach and intestines, is so difficult that, practically, from an etiological and clinical standpoint, they go hand in hand.

The Age. Diseases of the liver usually occur late in life because the causes upon which they depend are operative only at that period of life. In a case, therefore, of ill health in a young subject, the cause of which cannot well be determined, the liver is not so likely to be the seat of disease as in older subjects. Late in life we have the occurrence of

gall-stones with their multiple consequences, of cirrhosis, and of cancer. We may have in early life, although not so frequently, the congestions and the degenerations.

The Sex. The sex is not of much significance from a diagnostic standpoint. Cancer may be more frequent in the female sex, because cancer of the uterus and other organs is more common. Cancer of the biliary passages is more frequent in females, because in that sex gall-stones, which are etiological factors in cancer, are more common. Cirrhosis also is stated to be relatively more frequent in females.

The Habits. It is always necessary to inquire into the habits in order to determine the diagnosis. Alcoholism points to cirrhosis; the excessive use of stimulating foods to hyperæmia; sedentary habits and the use of starches and fats to gall-stones. The occupation has but little influence in the development of hepatic disease. With regard to climate it may be said that in tropical countries hyperæmias and abscess of the liver are more frequent.

Previous Disease. It is absolutely essential to inquire into this to establish a diagnosis. The occurrence of heart disease or obstructive lung disease points to a congestion; infectious diseases to cirrhosis, when it cannot be accounted for otherwise; dysentery to abscess; ulceration or suppuration in the portal area to multiple abscess; syphilis to syphilitic disease; tuberculosis, suppurations, bone disease, and syphilis to amyloid disease; pyæmia to multiple abscesses; tuberculosis to fatty liver.

The Subjective Symptoms.

The subjective symptoms are such as belong to functional disorder of the liver, conspicuous among which are gastro-intestinal symptoms and toxæmia. (See Functional Disturbance and Lithæmia.)

Pain is a frequent symptom of liver disease. When sudden in onset, acute, and increased by pressure or movement, it is due to perihepatitis. Acute paroxysmal pain below the ribs points to gall-stones. It may be in the seventh or eighth interspaces. The paroxysms may occur at varying intervals and are often attended by jaundice. Pain with distention occurs in congestion. Stabbing or darting pains occur in cancer. The pain of perihepatitis may attend abscess.

Pain in the liver must not be confounded with pleurisy. In pneumonia there is often congestion of the liver and perhaps perihepatitis. The pain has been taken for the pain of hepatic colic.

The Data Obtained by Observation. The Objective Symptoms.

Topographical Anatomy. The right lobe of the liver is applied to the concavity formed by the lower lobe of the right lung, being separated from it by the diaphragm. The thin lower edge of the right lung overlaps the liver at its upper part, but the greater portion of the anterior surface of the right lobe of the liver is in contact with the ribs. The under surface of the liver is in relation with the stomach, transverse colon, duodenum, right kidney, and right supra-renal capsule. "The highest part of its convexity on the right side is about one inch

below the nipple, or nearly on a level with the external and inferior angle of the pectoralis major. Posteriorly the liver comes to the surface below the base of the right lung, about the level of the tenth dorsal spine." (Holden.)

Roughly speaking, the upper border of the liver corresponds with the level of the tendinous centre of the diaphragm, that is, the level of the lower end of the sternum. Thus a needle thrust into the right side, between the sixth and seventh ribs, would traverse the lung, and then go through the diaphragm into the liver.

The attachments of the liver permit of a certain amount of movement. Hence the liver can be depressed by deep inspiration, emphysema of the lungs, or right pleural effusion. If the patient lie upon his left side the left lobe of the liver rises higher and the right extends lower, and *vice versa* if the patient lie upon the right side; the liver turning upon the suspensory ligament as an axis. (Gerhardt.)

Inspection. Inspection is not of very great assistance in the diagnosis of diseases of the liver. Frequently there is a swelling in the right upper quadrant, which may or may not be produced by an enlargement of the liver, but which should direct attention to that organ. The lower right zone of the thorax may also be distinctly prominent. Such a swelling may be observed in amyloid disease, hydatid tumor, cancer, abscess, and less frequently in fatty liver. In amyloid and fatty livers the projection in the right upper quadrant, which may extend to the left beyond the median line, presents a smooth surface, whereas in *hydatid tumor* there is frequently a rounded projection at some part of the prominent area, and in *cancer* several nodules may be large enough to cause slight rounded projections, which the eye is more apt to detect after the sense of touch has first directed attention to their presence.

Enlargement of the superficial abdominal veins on the right side is a common accompaniment of cirrhosis.

JAUNDICE. The color of the skin and of the mucous membranes which takes place in jaundice has been described (see page 71). In addition to the yellow discoloration jaundice causes a number of symptoms: 1. *Irritations of the skin.* Pruritus is common and intense, and may cause great distress. An attack of jaundice may be preceded by general itching. It occurs in all forms, but is more marked in obstructive jaundice of long duration. Scratch-marks are seen on the surface of the skin, and erythematous eruptions and boils frequently occur. *Xanthelasma* is a peculiar affection occurring on the tongue, on the skin of the eyelids, and about the ears (see page 123). 2. *Discoloration of the secretions.* All the secretions of the body are changed in color, as previously described. 3. *Bile absent in the fæces.* The stools are ashy, or gray in color. 4. *Slowness of the pulse.* The heart's action falls to 40 or 30 to the minute, or even lower. 5. *Hemorrhages.* In the later stages of all forms of jaundice hemorrhages are of common occurrence. In acute malignant jaundice they are seen underneath the skin, and occur from the mucous membranes. 6. *Cerebral symptoms,* irritability, and depression of spirits are marked. As the disease advances mental acts become sluggish; the patient is dull, and sleeping most of the time. Gradually the symptoms of the

typhoid state develop. In the acute febrile forms, coma and convulsions follow this condition. In the affection known as *acute yellow atrophy* the cerebral symptoms are marked, and occur early. Within the first twenty-four hours there may be convulsions, with delirium in the intervals, and subsequently coma.

Causes. Jaundice is (*a*) *hæmatogenous or non-obstructive* when (1) the function of the liver cells has been suppressed, as in acute yellow atrophy of the liver; (2) when blood destruction is in excess of the capacity of the liver to remove the products of destruction, the bilirubin, as in certain forms of malaria, in pernicious anæmia, in certain fevers, and other toxæmias; (*b*) *hepatogenous* when there is obstruction of the ducts. The obstruction may take place in the large ducts or in the smaller terminal ducts. The obstruction is due (1) in the large ducts, to disease outside of the ducts; (2) in large and smaller ducts, to disease of the ducts; or (3) in all sizes, to obstruction within the ducts. Hence we have jaundice.

1. From the pressure upon the ducts, of tumors connected with the stomach, kidney, pancreas, or the omentum; of tumors of the liver itself, or enlarged glands in the fissure of the liver; of accumulated fæces in the colon; of abdominal aneurism; and in rare instances, of the pregnant uterus.

2. From catarrhal inflammation of the mucous membrane of the ducts; suppurative inflammation of the same; adhesive inflammation of the ducts; cancer or other tumors at the orifice, or within the duct.

3. From foreign bodies within the ducts, as inspissated mucus, gall-stones, or parasites.

Diagnosis. Jaundice due to *disease outside of the ducts* is gradual in onset, varies in degree with the extent of pressure, becomes chronic, except in pregnancy and from fæcal accumulation; may cause a fatal termination, or persist until such termination results from the primary disease. It may be recognized by the absence of pain; the presence of disease in other localities, indicated by the symptoms and signs thereof; the absence of a history of gall-stones; and finally, the age of the patient. In the large majority of cases this form of jaundice is due to *disease of the pancreas*, particularly carcinoma.

Jaundice due to *disease of the ducts* presents varying features. The most common form is that due to *catarrhal inflammation* of the ducts. The jaundice comes on suddenly, at least within forty-eight hours after the onset of the symptoms; it occurs without pain, and is attended by vomiting and other symptoms of mild gastritis. The jaundice is usually attended by itching. It follows indiscretions in diet, and occurs in young subjects. Generally a pronounced cause for the gastritis can be ascertained. If the jaundice is due to *suppurative inflammation of the ducts* there is a history of gall-stones preceding, on account of which the suppuration took place. It must not be forgotten, however, that other lesions which cause jaundice may cause suppurative inflammation of the ducts also, such as obstruction by external pressure. The course of the jaundice is chronic. Fever and other symptoms of suppuration attend it. In *adhesive inflammation* there is a history of trauma from gall-stones, and the affection is chronic. In *cancer of the*

gall-ducts the advent of the jaundice is slow, the course protracted; the symptoms are the symptoms of carcinoma, to which are often added the symptoms of suppuration. (See Disease of the Gall-ducts.)

Foreign bodies within the ducts cause jaundice by direct obstruction, or because of the catarrhal inflammation which their presence excites. The symptoms occur suddenly in the former instance, gradually in the latter. The characteristic symptoms of gall-stones precede the jaundice. The patient is usually a woman past forty with habits of life which predispose to the formation of calculi.

Jaundice due to lowering of the blood pressure in the liver, so that the tension is altered between the bile-ducts and the blood passages, occurs suddenly, is light in degree, and is not attended by marked symptoms of jaundice; it is due usually to shock or depressing emotions.

Hæmatogenous jaundice must be distinguished from hepatogenous jaundice. In the hæmatogenous form the onset of the jaundice is more rapid, the general symptoms that attend it are more pronounced, particularly the nervous symptoms. With the onset of discoloration cerebral symptoms are observed. This is particularly the case in acute yellow atrophy of the liver. In the toxic forms of hæmatogenous jaundice in which there is no obstruction the symptom is not severe; the discoloration of the skin is light yellow; it may not be observed by the patient, and does not cause pronounced symptoms. The blood is destroyed rapidly in these cases, and as it cannot be disposed of by the liver, spleen, or kidneys, the transformed hæmoglobin is deposited in the tissues. In this class of cases the urine contains but little bile pigment, but there is a large amount of bilirubin and indican. The stools are not clay-colored.

Infantile Jaundice. Jaundice in infants is due to two causes: First, congenital obliteration of the ducts; and, second, catarrhal inflammation. It must not be confounded with the yellow discoloration of the skin due to the excess of coloring matter of the blood which is not disposed of by the liver. In congenital obliteration of the gall-ducts jaundice rapidly ensues, and deepens to an intense degree; hemorrhages occur, the child becomes stupid or comatose, may have convulsions, and death takes place in coma. There is rapid emaciation, and the liver and spleen are enlarged. The child may live many months.

Simple catarrhal jaundice in infants is associated with moderate gastric disorder. The jaundice is light; the conjunctiva alone may be discolored. In infants malignant jaundice may be due to inflammation of the portal veins secondary to umbilical phlebitis. The jaundice develops after local inflammation about the umbilicus, in which a slight puriform discharge is seen at the navel, attended by an increase in temperature. There may be some tenderness over the liver; frequently peritonitis develops at the same time. Pyæmic symptoms may set in, and pus be found in other situations. If the fever and pyæmic condition do not cause death the jaundice becomes more pronounced, and causes cutaneous and mucous hemorrhages. Convulsions and coma are apt to supervene before death. Jaundice in infants also occurs in interstitial hepatitis of syphilitic origin. The evidences of hereditary syphilis are seen in the skin and mucous membranes. The liver is enlarged, and there may be tenderness on account of perihepatitis.

Malignant Jaundice. *Acute Yellow Atrophy of the Liver.* Acute diffuse inflammation of the liver with necrosis of the cells, characterized by jaundice and cholæmia. It occurs very frequently in females during pregnancy. It is most common prior to the thirtieth year of age. It is said to follow fright. The symptoms are local and general. Jaundice at first is noticed coming on after an attack of gastro-duodenal catarrh. It is light, occasionally extends over the entire body, is not usually attended by itching. Within twenty-four or forty-eight hours the patient complains of headache; delirium sets in with stupor and the occurrence of convulsions. With the onset of the headache vomiting takes place. Fever of moderate degree begins at the same time, although in some cases it is absent. Although the jaundice is not intense, the effects upon the blood are early seen. Hemorrhages underneath the skin and from the mucous membrane take place. In pregnant women abortion follows, the hemorrhage from which may be very excessive. The stupor and delirium are followed by coma, and death takes place within a week of the onset of the disease; or coma may be preceded by the typhoid state, and the disease last for a week or more. The urine is bile-stained, and contains albumin and casts. It diminishes in amount, and is soon passed involuntarily. Leucin and tyrosin are always present. The latter may be seen in the sediment, although it is more marked when a few drops are evaporated on a cover-glass. The bowels are loose and the stools involuntary and clay-colored.

On examination of the liver the organ is found to be diminished in size; this may not be appreciated by percussion in the anterior region, but in the axillary region the width is reduced one or two inches. There may be some tenderness over the liver and over the ducts. The data upon which a diagnosis is based are the age, sex, occurrence of pregnancy, the rapidity of onset of cerebral symptoms following jaundice, diminution in the size of the liver, with leucin and tyrosin in the urine. It must be distinguished from the jaundice that attends hypertrophic cirrhosis of the liver, which at times becomes malignant. Some observers have thought that necrosis of cells had supervened upon this lesion, but fever is more marked in this form of jaundice, and leucin and tyrosin are absent from the urine.

It must not be forgotten that all cases of jaundice may terminate suddenly with delirium, followed by coma, or by the development of the typhoid state. In phosphorus poisoning the hemorrhages, the jaundice, and diminution in the size of the liver are the same as in acute yellow atrophy. Gastric symptoms are more marked, and leucin and tyrosin are not present in the urine.

Fever. *Hepatic Fever.* In addition to the determination of the cause of jaundice by the character of symptoms and the associate phenomena, the occurrence of fever may be of diagnostic importance in distinguishing the various forms of obstructive jaundice. Fever occurs frequently in jaundice, but usually attends only certain forms. In catarrhal jaundice it is present for three or four days only, disappearing as the severe gastric symptoms subside. In hepatic colic it is transitory and associated with chills and sweats. In jaundice from obstruction

it occurs, first, when the obstruction is due to gall-stones without secondary changes in the liver; second, in suppurative inflammation of the ducts produced by the stone or from other causes. Fever under these circumstances assumes a peculiar form which, on account of its association with disease of the liver, is known as intermittent hepatic fever (see p. 114). The fever is associated with obstructive jaundice in the following groups: First, with each paroxysm of hepatic colic both fever and jaundice are present. The latter, becoming more intense after each paroxysm, may persist for months or years. Second, jaundice persists, and is attended by distinct ague-like paroxysms of chill, fever, and sweat, after each of which the jaundice is more intense. Third, pain in the liver and gastric disturbance, with fever, but without jaundice. The pain and gastric disturbance occur in distinct paroxysms. *Gall-stones* are probably the cause in all these conditions, leading in some to chronic obstruction of the duct without suppuration. If suppuration is present the symptoms are somewhat different. Thus, (1) there is more tenderness in the hepatic region, with enlargement of the gall-bladder; (2) paroxysms are more frequent in suppurative inflammation; (3) jaundice is not so intense and not influenced by paroxysms; (4) in suppurative inflammation the patient is ill in the intervals, and there is wasting. There are are no periods of improvement locally or in the general condition. The most important point is the comparative ease in the intervals between the paroxysms of fever in the case of gall-stones.

Intermitting fever of this character must be distinguished from malaria. The history of gall-stones, with pain in the region of the liver, and the negative appearance of the blood, are sufficient to point to the diagnosis.

Fever in disease of the liver also occurs in cancer when the neoplasms grow rapidly, in certain forms of cirrhosis, and in obstruction from other causes than gall-stones. It is particularly common in suppurative inflammation of hydatid cysts, or after they rupture and discharge into the biliary vessels. Without previous knowledge of the hydatid cyst the diagnosis is almost impossible, save that the pain is less when obstruction is due to this cause than in obstruction from the passage of gall-stones.

Weil's Disease. *Acute febrile jaundice*, which rapidly becomes malignant, occurring in butchers, laborers, and brewers, has been described by Weil. After exposure to cold generally, as in a beer vault, the patient is seized with a chill, followed by fever, with headache, vomiting, and epigastric pain. Jaundice sets in rapidly. The temperature remains high and may be intermitting. Stupor, delirium, and coma, albuminuria with suppression of urine, subcutaneous hemorrhages, and hemorrhages from mucous membranes rapidly ensue. Black vomit occurs early. In one of my cases there was enlargement of the liver with œdema over the surface. The microscopical appearances were those of acute diffused parenchymatous inflammation. In another, a breweryman, the liver was enlarged, but without unusual change, save congestion.

The delirium is sometimes violent. The appearance and symptoms

suggest acute yellow atrophy of the liver. The ætiological distinctions are noteworthy: the liver is not small; leucin and tyrosin are not found in the urine; the jaundice is more intense. The diagnostic circumstances of epidemic and contagious diseases serve to exclude yellow fever. (See Yellow Fever.)

Palpation. By palpation the lower border of the liver can be determined in thin subjects or in those in whom the liver is greatly enlarged. It may be difficult to determine the border when the abdomen is distended on account of flatulency. Careful palpation must be made with the tips of the fingers, firmly pressing them inward along the margin of the ribs, at the same time securing relaxation of the abdominal muscles by having the patient take a full breath, and having the legs drawn up and the shoulders elevated. The pressure should be made in the intervals following the act of inspiration. By care and patience the fingers can be pushed deeply inward and be made to feel the border of the liver, even in health. Care must be taken not to cause contraction of the right rectus muscle, for if this takes place the indurated mass may simulate tumor or enlargement of the liver. The left lobe of the liver below the ensiform cartilage extends half-way to the umbilicus. Here it is most accessible to palpation. By palpation we also determine the size of the gall-bladder and the degree of movement of the liver in respiration. On full inspiration the liver descends, and during the act of expiration rises again. This movability is of service in determining the liver from other organs that are fixed within the abdomen.

In *amyloid* disease the lower edge is smooth, rounded, the tissue dense and unyielding to pressure, and the anterior surface perfectly smooth, as a rule; but when the liver is also cirrhotic or syphilitic, the surface may be irregular and fissured.

The *fatty* liver has also a rounded smooth border, but its tissue is not so dense and resistant, except when cirrhosis coexists. Its surface is smooth.

In single *abscess* the liver is enlarged, but not uniformly, and not invariably. If the abscess is located in the right lobe and nearer the anterior than the posterior surface, palpation may be able to detect not only enlargement but also deep-seated obscure fluctuation, surrounded by a zone of hard tissue. The tumor is round, smooth, tense, tender and painful.

In *multiple abscesses* the liver is enlarged uniformly, and usually none of the abscesses are large enough to be felt as a distinct prominence. The liver is tender and painful.

In *hydatid tumor* the degree of enlargement depends very much upon the situation of the cyst, upon its stage of development, and upon the activity of the echinococci. Sometimes the cyst is so small that its existence remains unsuspected; at other times the enlargement is so great as to fill the abdominal cavity. As in abscess, the possibility of detecting the tense, globular, fluctuating, painless tumor characteristic of the disease, depends upon its situation. If upon the anterior surface or lower border, this is very easy, especially if the tumor is at all large; but if it projects from the posterior surface or from the upper or lateral borders detection of the tumor is difficult, and may be impossible.

In *congestion of the liver* the enlargement is not so great as in abscess, nor are pain and tenderness so pronounced. Moreover, the enlargement is usually not permanent. The lower border, as it projects below the edge of the ribs, is smooth.

In *hypertrophic cirrhosis* the enlargement is moderate, the surface smooth or but slightly roughened, denser than normal, and somewhat tender.

In *cancer* the enlargement resembles that of single abscess and hydatid tumor in that it is irregular. But, unlike hydatid tumor, the irregularities are due to knobs which project from the surface of the liver, are usually entirely free from any fluctuation, and are tender on palpation. There may be a single large mass, or a number of knobs or nodules. The part projecting below the ribs may be free from any nodules.

Palpation of the liver may discover a *friction* from perihepatitis, and *pain or tenderness* from that cause, cancer or abscess. *Pulsation* of the liver may be a transmitted impulse from the abdominal aorta or a venous pulse, such as occurs also in the jugulars, from tricuspid regurgitation.

Floating liver is diagnosed by feeling in the lower, most frequently the right portion of the belly, a large tumor which can easily be confounded with tumors of other organs. It can be distinguished as liver: (1) By recognizing the notch; (2) by the presence of a tympanitic note in the proper region of the liver, as loops of intestine lie between the diaphragm and liver; (3) by excessive movability of the tumor; and (4) by ability to replace the liver in its proper position. It occurs almost invariably in women, probably as the result of a congenital lengthening of the suspensory ligament.

Constriction of the Liver from Tight Lacing (*Schnurleber*) occurs especially in women. Tight corsets and still more tight waist-straps or bands squeeze the liver downward, especially the right lobe, so that it can be palpated. In more pronounced degrees of the condition a furrow, often palpable, is produced, and below this a constricted lobe which may extend as far down as the anterior superior spine of the ilium and carry the gall-bladder with it.

Lobes so depressed are usually thin and easily movable, and can be grasped with the hand and moved to and fro. If the lobe does not reach so far downward it is more rounded and blunt in shape. It is not always easy to demonstrate its connection with the liver, because coils of intestine lie over the liver in the furrow, make palpation difficult, and introduce a tympanitic note between the liver dulness and the dulness of the constricted lobe.

Confusion with tumors of other kinds can be avoided usually by deep palpation or percussion.

GALL-BLADDER. When the gall-bladder has a certain degree of fulness, it may, according to Gerhardt, be not only felt in healthy persons, if the stomach and bowels are empty, as a smooth, round, fluctuating tumor at the lower border of the liver, but be even visible and be outlined by percussion. If a line is drawn from the right acromion process to the umbilicus, it will bisect the gall-bladder at a point where

it passes over the margin of the ribs. The fundus is situated below the edge of the liver, at about the ninth costal cartilage, just outside the edge of the right rectus muscle. Palpation is easy when, owing to closure of the cystic duct, the gall-bladder is distended with bile or with inflammatory exudate, or enlarged by thickening of its walls or by an accumulation of gall-stones. A pear-shaped tumor is then felt which, if not adherent to the border of the liver, is shoved up and down with it. In simple stasis, hydrops vesicæ felleæ, and purulent inflammation, the tumor is tense and elastic; in inflammatory or carcinomatous thickening of the wall, dense and irregular. Calculi can often be recognized by their form or hardness or by the sound made by rubbing them together.

Aspiration. We are warranted in determining the nature of an obscure enlargement of the liver or of the gall-bladder by aspiration. In abscess, pus; in hydatid disease, the characteristic fluid, may be withdrawn.

In a case of local enlargement the apex of the swelling should be aspirated. If aspiration is performed near the upper border the needle should be thrust downward; if near the lower border, upward. The left lobe should be aspirated with care in order that the stomach be not pierced.

Percussion. *Alterations in Size and Shape of the Liver.* The liver may diminish in size or it may enlarge. *Diminution in size* can only be recognized by percussion. The normal extent of hepatic dulness is diminished. This is usually more marked in the anterior and lateral regions. It must not be confounded with the apparent diminution that takes place in emphysema, or that may occur from distention of the bowels with flatus, as in peritonitis. Absence of hepatic dulness may occur when there is gas in the peritoneal cavity. *Enlargement* of the liver is determined by inspection, palpation, and percussion.

By *percussion* the size of the liver is accurately made out. Any marked increase beyond the normal limits (see p. 590) usually means increase in size of the liver. Both superficial and deep percussion must be performed. The upper border is determined by percussing from a point beyond the liver area toward the liver—anteriorly from the third interspace downward, laterally from the fourth, and posteriorly from the angle of the scapula. In health the upper border of the liver is found at the fifth interspace; in the axilla, at the sixth; and in the back at the ninth interspace. From thence downward hepatic dulness should continue to the margin of the ribs. It falls short of this position by at least an inch in the aged, and in persons with a deep chest it may in front not be more than two inches in width. The width of the liver dulness in the right mid-clavicular line is about four inches, in the mid-axillary line six, in the mid-scapular line three inches.

The enlargement may be uniform, it may be limited to one lobe, or it may be irregular. By percussion it may be found that the enlargement is regular from increase in size upward or downward, or increase in the area of dulness in both directions. On the other hand, the enlargement may be irregular. The liver dulness may begin higher in the anterior region than in the axillary region, or may extend downward over the

margin of the ribs in a circumscribed area. Sometimes the enlargement is limited to the left lobe and the increase in size noted by increase in the dulness from the xiphoid cartilage downward as far as the umbilicus. The entire middle region to the navel may be filled up by the enlarged liver.

Uniform enlargement of the liver is due to congestion, fatty degeneration, amyloid disease, cancer of the liver, and sometimes to hydatid disease and abscess of the liver. *Enlargement of one lobe* of the liver is due to hydatid disease, to abscess, or to cancer, in nearly all cases. Either the right or the left lobe may be the seat of such enlargement.

Enlargement in one particular direction is due also to the three conditions just indicated. Although enlargement downward by abscess or hydatid disease is the more common one, the enlargement may be directly upward, the lower border of the liver occupying the normal position. Enlargement of the liver upward is due to a cyst, or an abscess, which has developed in the convex surface of the right lobe.

Irregularity in the shape of the liver dulness occurs in cancer, in abscess, and hydatid disease. Notwithstanding the apparent irregularity, enlargements of the liver always occupy the *normal site* of the organ and conform to its usual outline, with but moderate variations only.

Enlargements of the liver must be distinguished from enlargement of organs in contiguity with the liver, or from structures usually containing air, which have become solid or non-resonant structures. The enlargement must therefore be distinguished from pleural effusion, or disease of the lungs which causes dulness on percussion, or from disease of the abdominal organs on account of which there is increased dulness near the hepatic region. Hence, in renal tumors, in tumors associated with the large intestines or stomach, in ovarian tumors, in tumors due to accumulation of fæces, the physical signs on percussion may show their similarity to enlargement of the liver.

Simulated Enlargement. It is well to bear in mind the conditions which simulate enlargement of the liver. Of these we have: (1) *Congenital* malformation; the liver may be of abnormal shape, on account of which the area of dulness will be increased in a particular direction. It may be quadrangular or rounded. The liver may be found in the right pleural sac in congenital diaphragmatic hernia. The increase of dulness upward will simulate enlargement of the liver. Congenital malformations may be suspected in the absence of any symptoms of hepatic disease, or of conditions which may cause other forms of spurious enlargement. Moreover, the increased dulness will have existed from early life. (2) In *rhachitis* on account of the malformation of the chest, the position of the liver may be such that its area will increase every way. For the same reason the liver may be felt below the margin of the ribs. (3) Disease of the *spinal column* causes dislocation, on account of which the liver may appear to be apparently increased in size. (4) Enlargement of the liver must be distinguished from *pleural effusions*. This is sometimes difficult. The symptoms of the pulmonary affection must be considered. The general conditions which cause hydrothorax must be borne in mind. The difficulty in distinguishing the two occurs because the dulness of each is continuous. In the pleural effusion, however, there

is uniform bulging of the affected side. The liver is not movable, the chest expansion is lessened. The upper border of dulness of the fluid may be movable if the effusion is not large. If the effusion is small the line of dulness is S-shaped. It is high behind and high in front. If the effusion is large the upper limit of dulness is horizontal. The upper limit of dulness in the pleural effusion changes its position in many instances. In enlargement of the liver the lower costal ribs are often everted, but in pleural effusion a depression may be seen between the lower margin of the ribs and the upper surface of the liver, if the latter is dislocated by pressure of the fluid. Sometimes enlargements of the liver give rise to secondary pleural effusion, so that too often after finding pleural effusion the size of the liver is not estimated. (5) *Pericardial effusion* and dilated heart are said to simulate enlargement of the liver. The history of the case, the origin and mode of development of the symptoms, the physical signs of cardiac disease, point to its true nature. (6) Enlargement of the liver may be due apparently to *subdiaphragmatic abscess.* The accumulation between the liver and diaphragm causes the latter to be pushed downward. It is very difficult to distinguish the spurious from the false in these instances. *Aspiration* may help in the diagnosis. (7) *Abnormal Conditions of the Abdominal Parietes.* Increased tension or spasm of the recti muscles. Phantom tumors of the abdomen simulate enlargement of the liver. They occur in young girls, associated with gastro-intestinal catarrh and symptoms of hysteria. Anæsthesia must often be employed to disperse the phantom.

(8) *Tight Lacing.* This may displace the liver upward or downward, according to the direction of pressure. It may also, by exerting lateral compression, bring more of the liver into contact with the anterior abdominal wall. And finally, if the constriction has been by a strap or tight cord, a portion of the liver may be more or less detached and appear as a movable tumor.

(9) Some enlargements of the *abdominal contents* cause spurious enlargement of the liver. In the same way increased abdominal pressure (ascites, tympanites, etc.) causes the liver to rise higher than normal.

a. The accumulation of fæces in the colon. This causes continuance of liver dulness downward, on account of which it may be thought that the patient has liver disease. A purgative must be given.

b. An ovarian cyst.

c. The presence of ascites. Exclusion of the latter is sometimes difficult because the ascites may be loculated and situated in the hepatic region. It may give rise to symptoms of hepatic enlargement. Probably aspiration alone can make the diagnosis distinct. Ascites should be easily distinguished by the physical signs and the results of exploratory puncture.

d. Tumors of the omentum, chiefly tuberculous, may occupy such relation to the liver as to increase the dulness downward. The history, the occurrence of the omental tumor, with symptoms of tuberculosis, may aid in determining the true condition.

e. In tumors of the kidney which simulate enlarged liver it is found that the edge of the liver cannot well be felt, but Murchison thinks the fingers can usually be inserted between the ribs and the upper part of the

renal tumor. The renal tumor, however, is not fixed. It is rounded on every side; it partakes of the shape of a kidney. The urine should be examined.

f. Enlargements of the liver must be distinguished from pancreatic cyst, or effusion in the lesser peritoneal cavity. This can usually be accomplished with ease, except in hydatid disease of the left lobe near the suspensory ligament. In effusion of the lesser peritoneal cavity the tumor occupies the left upper quadrant, and may extend as low as the transverse umbilical line. It causes dislocation of the heart, so that the apex is as high as the third interspace, and beyond the mid-clavicular line. It is accompanied by an increase in the dulness posteriorly, so that the upper limit may extend to the angle of the left scapula. The results of puncture alone may be sufficient to distinguish them.

A clue to the nature of enlargement of the liver may sometimes be formed by the presence or absence of pain. Murchison makes this a reliable distinction. Painless enlargements of the liver are due to congestion, to hydatid disease, to fatty and amyloid disease of the liver. Painful enlargements of the liver are seen in abscess, cancer, and syphilitic disease, with perihepatitis.

In childhood the lower border of the liver normally is lower than in adults, because the liver is itself proportionately larger than it becomes later. For the same reason the upper border is at a higher level.

Diseases of the Liver. The Fatty Liver.

The symptoms of fatty liver are not pronounced. The physical signs are those of enlargement, which is uniform and extends in all directions. On palpation, the edges can be felt; they are rounded and smooth. They are soft at first, but later become indurated. Fatty liver may be followed by cirrhosis after a period of alcoholism. The general symptoms are those of the primary disease. Fatty liver occurs in gouty subjects, but is notably present in wasting diseases, in tuberculosis, in chronic hip-joint disease, and in amyloid disease of the liver.

Fatty liver sometimes follows the congestion of the liver which is present in the course of organic heart disease. The liver is not truly fatty, but properly should be called a fatty cirrhosis. There is increased fatty degeneration with an overgrowth of connective tissue. This form is associated with heart and kidney disease. On palpation the edges of the liver are hard or indurated. The liver may undergo diminution in size later, and the symptoms of cirrhosis ensue.

Amyloid Disease of the Liver.

Disease of the liver attended by enlargement without pain, is often due to amyloid disease. Similar disease is found in other organs, and there is present, to point to the nature of the enlargement, bone disease, prolonged suppuration, or tuberculosis. In amyloid disease the pallor of the patient is pronounced and the face may be swollen, and the ankles slightly œdematous. The spleen is enlarged, the urine albuminous, scanty, but of moderate specific gravity. In amyloid disease

a history of syphilis is an important point in establishing the diagnosis. Fatty liver can readily be distinguished from amyloid disease by palpation. In amyloid disease the surface is smooth, but it is very hard and indurated.

Cancer of the Liver.

The ætiological factors upon which the diagnosis of cancer is based are: the age of the patient—most frequently between the fortieth and sixtieth year; the female sex, in a measure, and heredity. The disease is nearly always secondary to cancer in some other situation, consequently in cases in which symptoms point to cancer of the liver search must be made for the primary lesion elsewhere. Of these the most frequent are the rectum, the uterus, the stomach, the remainder of the gastro-intestinal tract. Cases have been reported in which the eye has been removed for unrecognizable disease, and symptoms of carcinoma of the liver have subsequently developed. The nature of the hepatic symptoms was obscure during life, but at the post-mortem examination melanotic sarcoma was found; the primary lesion was undoubtedly present in the eye. Further ætiological influences that may bear upon the diagnosis are: 1, the occurrence of gall-stones, which act as the exciting cause in the development of primary cancer of the ducts, from thence spreading to the liver; 2, the occurrence of trauma.

The *symptoms* of cancer of the liver are due to increase in size of the liver, to pressure of the growths upon the ducts or terminal portal vessels; and to the general effects of carcinoma upon the system. The liver is enlarged and its surface irregular. The organ can be made out by palpation extending below the margin of the ribs. The edges are irregular, and, on the surface, bosses can be distinctly felt. In rare cases one or two masses alone may be present, growing out of the substance of the left lobe of the liver, on account of which a large tumor may be seen below the sternum. The nodules are usually hard, but sometimes may be soft and even fluctuate. The abdomen is distended.

After emaciation becomes marked the nodules can be seen near the surface of the skin, and even the multiplication distinctly made out.

The liver is movable with each inspiration; the enlargement can be noted while under observation. By percussion the enlargement can be distinctly made out, and while the surface is irregular, the general shape of the dulness corresponds to that of the liver. On account of the increase in size and of inflammation of the capsule the patient complains of weight in the hepatic region, and of pain which may be intermitting in character. The nodules may be tender on palpation. The superficial veins are enlarged.

In not every instance do we find enlargement. In some cases the cancer is associated with cirrhosis of the liver or may itself be of a nodular type which shrinks. The liver is then normal or diminished in size, as indicated by percussion.

The symptoms that attend cancer are: 1. Jaundice, which is not very deep unless the common duct is affected. 2. Ascites, which is always

present in the atrophic forms, but may be absent when the liver is enlarged. 3. The general symptoms are those of emaciation, which is rapid; of prostration and, in some instances, of fever. Fever of moderate degree attends the rapidly growing cases. It is usually continuous, but may be intermittent, especially if there is suppuration, or suppurative inflammation of the ducts. It is a well-known fact that gall-stones are of common occurrence in patients the subject of cancer in whatever location it may develop. The symptoms of biliary calculus or of obstruction may attend those of secondary cancer of the liver, and the stones bear an ætiological significance.

In many instances secondary cancer of the liver may be present without symptoms during life to attract attention to this organ. If cancer in other regions has continued for the usual period of time, it may be almost certain that at the autopsy cancer of the liver will be found to be present.

Diagnosis. The diagnosis of cancer of the liver is not difficult when the changes in the liver can be made out on palpation and percussion. In rare instances in which the liver is smooth it may be mistaken for fatty or amyloid liver. A definite cause can usually be made out for the latter, while the occurrence of jaundice, the rapid increase in size of the liver, and the general symptoms of the cancerous cachexia indicate the hepatic affection. The *syphilitic liver* with irregular gummata may cause serious doubt; the history of the case and other signs of syphilis aid in the diagnosis. Locally the condition may exactly simulate carcinoma. The jaundice, however, is not so frequent in occurrence or so deep in syphilitic gummata; the cachexia does not ensue, but the therapeutic test may be essential in order to make a diagnosis.

In *hypertrophic cirrhosis* of the liver the jaundice is deep and the liver enlarged; there is but little wasting and anæmia. In this form of cirrhosis the surface of the liver is smooth; certainly there are not any bosses, and the organ is painless. Ascites is more common in cirrhosis; the patient is usually affected earlier in life than is true in cancer.

In a large growing cancer one or two of the nodules may suppurate and simulate *abscess of the liver.* In the absence of cause for the abscess, the age of the patient and the results of aspiration favor carcinoma. If the cause is dysentery, the age, early life, occurrence of persistent pain, irregular enlargement of the liver, the development of anæmia, but not of cachexia, and very marked hectic, without jaundice, favor abscess. If the enlargement is posterior it may set up localized pleuritis or moderate pleural effusion, as rarely occurs in abscess.

Cancer of the liver may be simulated by cancer of organs in close proximity to the liver, as the *pancreas*, the *pyloric* end of the stomach, or the *colon.* In pyloric cancer the symptoms of dilatation of the stomach are present; the percussion note is not dull, but there is a dull tympany over the tumor; it is attended by vomiting and possibly hemorrhage from the stomach. Jaundice occurs late. Cancer of the pyloric end is not movable with respiration unless it becomes adherent to the liver. *Cancer of the omentum* and *colon* are not modified by respiration. The

percussion note over them is different; they frequently extend beyond the liver confines and are associated with symptoms of obstruction of the bowels. *Fæcal accumulation* in the transverse colon must not be mistaken for cancer of the liver. The large masses adjacent to the liver may closely simulate cancerous nodules. Careful percussion must be resorted to to prevent confusion. Cancer of the liver and hydatid disease must not be confounded. The tumor in *hydatid disease* is usually single; it is large, and may fluctuate or yield the hydatid fremitus. It causes enlargement of the liver, the shape of which is irregular when the tumor presents in the epigastrium or along the margin of the ribs. It is painless. Aspiration yields the characteristic hydatid fluid.

Cancer of the bile-ducts cannot always be distinguished from cancer of the liver; although the occurrence of early jaundice in the patient the subject of gall-stones, with secondary enlargement of the liver and gall-bladder, at first smooth and painless, afterward irregular and painful, may point to the true nature of the case, particularly if a primary nodule cannot be found elsewhere. In cancer of the pancreas there is also difficulty; the occurrence of vomiting and of tumor in the mid-costal region, with the development of jaundice early, before the liver is enlarged or nodular, with other symptoms of cancer of the pancreas, as intestinal dyspepsia and fatty stools, may point to the primary lesion in this organ.

Cirrhosis of the Liver.

A diffuse interstitial inflammation of the liver, chronic in duration, usually with atrophy of the organ, is caused by irritants, in the large majority of cases, which enter the portal circulation through the stomach. Of the irritants alcohol is the most common, and particularly the stronger liquors, as gin and whiskey. Other irritants, as spices used to excess, may likewise cause the diffuse inflammation. In addition, however, cirrhosis of the liver may follow the infectious diseases, notably scarlatina, and may be incited by malaria. These forms of cirrhosis lead usually to atrophy of the liver.

Another form is due to obstruction of the bile-ducts with secondary overgrowth of the connective tissue. It is known as hypertrophic or biliary cirrhosis. In addition, cirrhosis of the liver may arise in the course of syphilis; the anatomical characters are different from those of true cirrhosis. This does not include an account of the secondary cirrhosis of the liver which arises in the course of a passive congestion of that organ, on account of which the so-called nutmeg liver develops.

Cirrhosis of the liver of the atrophic form, due to alcohol, presents various clinical features. In the first place it may exist without causing any symptoms whatever during life. It is to be found only after death from other causes, or it may not present symptoms until an accident in the course of the disease may occur, as hemorrhage from some portion of the collateral circulation. In both of these instances the symptoms are absent because the collateral circulation is complete. If this is incomplete, however, grave symptoms, local and general, ensue.

Before detailing them it may be well to state that the occurrence of one symptom which we have termed accidental may lead to the inference that cirrhosis of the liver is present, particularly if the patient has been an alcoholic. This symptom is hemorrhage. It may take place from the stomach, causing death at once, or after repeated hemorrhages have occurred; or it may take place from the intestine.

The Symptoms of Cirrhosis. The symptoms are general, due to interference with the nutrition of the patient; and local, dependent upon the degree of obstruction to the portal circulation. General symptoms rarely occur unless the local symptoms are present, which lead to malnutrition and mal-assimilation on account of interference with the gastro-intestinal digestion. In many instances a typical pattern of the disease is presented extending over a long period of time.

The symptoms observed throughout the disease have been referred to as symptoms of the first stage, or stage of enlargement, and symptoms of the second stage, or that of contraction. During the first stage the symptoms are those of gastritis, with enlargement of the liver. This so-called first stage is not always observed.

The gastric symptoms are those of morning retching or vomiting, with discharge of mucus, associated with other symptoms of gastric catarrh, as the loss of appetite, nausea, tenderness in the epigastrium, eructations, and constipation, with which symptoms the health may fail. The liver is *enlarged* and the outline regular. During the second stage more severe symptoms arise, due to obstruction of the portal capillaries.

The Signs of Portal Obstruction. The abdomen becomes distended and the sensation of weight and pressure is complained of. On examination *ascites* is detected. This may become enormous, causing monstrous distention with pouting of the umbilicus. The *spleen* is found to be enlarged, extending over twice or three times the normal area on percussion. If ascites does not interfere, the edge of the spleen can be readily made out. The portal obstruction causes secondary gastro intestinal catarrh, if it had not already been present on account of the alcoholism. Although constipation is usually present, there may be persistent diarrhœa. This may occur in the mornings only, and it may be lienteric. Hemorrhages may take place from the gastro-intestinal tract at any time either from the stomach or the intestine. Not infrequently they occur from the œsophagus, due to varicosity of the veins at the junction of the œsophagus and cardiac end of the stomach. Hemorrhoids are always present and may bleed at each stool. Jaundice is usually not the rule, and, if present, is usually light and generally due to the duodenal catarrh. The skin is of yellowish tinge or of a grayish-earthen color only.

Physical Examination. This may be rendered difficult on account of the large amount of ascites before paracentesis is performed. Early in the disease the enlarged liver of the first stage will be found to have undergone contraction, although diminution in the area of dulness is not by any means as absolutely confirmative of contraction as the opposite condition is of hypertrophy. Percussion should be performed several times, because the distended intestinal coils may modify the results.

Symptoms. The general symptoms of cirrhosis, and particularly the symptoms of the later stages, are striking and diagnostic. The nutrition is much impaired. The patient, who in the large majority of cases was at one time corpulent, becomes emaciated. The skin changes in color and becomes of an earthy-gray or dirty-sallow hue. The capillary venules of the face are dilated; the distended capillaries on the nose are distinct. Later, ecchymoses may occur in the skin and hemorrhages take place from the mucous membrane and into the retina. Debility ensues; œdema of the ankles is almost sure to occur, and sometimes general anasarca may take place. It is extremely rare to have fever unless complications occur. The pulse is small and becomes more rapid than normal; the heart sounds grow weaker. In the later stages the skin may be the seat of eruptions and chronic skin diseases of various kinds develop.

The *urine* throughout the disease presents no characteristics; as ascites develops it becomes scanty and dark, is loaded with urates and uric acid. In rare instances it may contain sugar, and if the uric acid is in excess, albumin.

With the distention of the abdomen enlargement of the superficial veins is also observed. This may be very pronounced, and particularly about the umbilicus. The enlarged swollen mass in this situation has been called, from its appearance, the *caput Medusæ.*

Collateral Circulation. The collateral circulation that develops in order that the portal blood may reach the right heart takes place in various ways. First, communication may be formed between the veins of the mesentery and those of the abdominal walls; second, between the coronary veins of the stomach and the veins of Glisson's capsule and the phrenic veins; third, between the internal hemorrhoidal and the hypogastric veins; fourth, enlargement of the obliterated umbilical vein in the ligamentum teres may take place.

In the study of a case of cirrhosis of the liver a judgment as to its nature may be in a measure confirmed by the presence of other phenomena due to the same cause. Very frequently we have at the same time cirrhosis of the kidneys and sclerosis of the arteries, with secondary atheroma, both of which have led to hypertrophy of the heart. Strümpell refers to the occurrence of cirrhosis and chronic tubercular peritonitis. He thinks the former is the primary lesion which predisposes to the development of the latter. The course of the disease is prolonged.

The duration cannot accurately be determined, as the onset is usually insidious. After the ascites appears the duration may vary from six to eighteen months. Of course this depends largely upon the degree of completion of the compensatory circulation. Death usually occurs from intercurrent disease or progressive exhaustion. In not a few cases cerebral symptoms occur. In addition to the cirrhotic cachexia, the sudden occurence of coma and convulsions, preceded by delirium, may ensue, the cause of which is not fully known. It must be borne in mind that the occurrence of these symptoms in an alcoholic subject may be due to a cirrhosis the presence of which had not been suspected during life.

Diagnosis. The diagnosis is usually not difficult if the complete picture of the case is presented. It cannot be established positively without definite knowledge of the cause. If the patient comes under observation after ascites has developed the diagnosis is more difficult. It must in the majority of cases be based upon exclusion of heart, lung, and kidney disease. The recognition of ascites of hepatic origin depends upon the history and the development of the disease. *Ascites* may be due to other causes within the abdomen. It may probably be difficult to exclude *chronic peritonitis*, which is a common cause of ascites. The general tenderness, the less degree of distention of the abdomen, and the absence of enlargement of the spleen point to peritonitis. The *fatty cirrhotic liver* may present symptoms similar to those of the atrophic form, except that it is enlarged at the time of the examination.

Hypertrophic cirrhosis, or so-called biliary cirrhosis, presents a somewhat different picture. In the first place the cause is different. It has usually been preceded by gall-stones, or by obstruction of the duct from other causes. The liver is enlarged, the surface is irregular. The induration of the uniformly enlarged liver is most striking. The enlargement causes weight and fulness, and is associated with weakness and loss of appetite. Jaundice very early ensues, or may have been the first symptom to call attention to the liver. It increases and persists throughout the course of the disease. Ascites is not usually present, or may be very slight. The enlargement and jaundice may continue for months or even years without the development of grave symptoms.

Fever may, however, set in at any time, in all probability generally due to the biliary obstruction. The fever is continuous; the temperature rises from 102° to 104°; the tongue becomes dry and brown; the pulse rapid. All the symptoms of febrile jaundice ensue. On the other hand, in the course of the disease the patient may be seized with convulsions, followed by coma and death. Most authorities state that the enlargement persists throughout the course of the disease, but other observers state that for a long period of enlargement with jaundice, if nervous symptoms or fever do not set in, contraction of the liver takes place, with subsequent development of the symptoms of portal obstruction. At this period the spleen may become enlarged and ascites take place, while the symptoms of digestive disturbances become more prominent. Nervous symptoms that arise may be due to acute diffuse necrosis setting in in the course of the disease.

The *diagnosis* is often difficult. Gradual and persistent jaundice without cause, continuing over a long duration of time, associated with persistent enlargement of the liver without symptoms of portal obstruction in the non-alcoholic subject, point pretty certainly to hypertrophic cirrhosis of the liver.

Syphilitic Disease of the Liver.

Syphilitic disease of the liver may result in cirrhosis on the one hand, or in the development of gummata. *Syphilitic cirrhosis* presents the same symptoms as the alcoholic form. The history, the more marked

irregularity on the surface or the edge of the liver, and the existence of syphilis elsewhere may lead to a diagnosis of the true condition.

In *congenital syphilitic disease* of the liver the inflammation is diffuse; the liver is enlarged and hard; the surface is smooth; there are usually syphilitic lesions in other organs; the patient presents syphilitic eruptions, and has the well-known wizened appearance that belongs to this affection.

Syphilitic gummata in the liver may exist without presenting any symptoms whatsoever, or their presence is known by the occurrence of pain and a localized swelling and discomfort, which calls the patient's attention to the region, particularly if at the same time the patient's health is reduced. Tumors are situated in the left lobe, in the median line, or along the margin of the ribs. Pain is usually localized to this region, but may extend over more or less of the liver, particularly if there is general perihepatitis along with other evidences of syphilis, which however may not be present. If the temperature is taken frequently a moderate febrile range will be observed. It may not rise above 100½°, but in the absence of other causes is a valuable diagnostic symptom. In other instances the gummata may grow in such a situation as to interfere with the portal circulation or press upon the gall-ducts. The latter is very rare. If the gummata are felt, they are enlarged bosses which give the sensation of flattened hemispheres. Sometimes on the surface of the enlarged organ several separate elevations can be made out. To determine the exact nature of the lesion is often very difficult. The symptoms may conclusively point to hepatic disease. Knowledge of the occurrence of syphilis or alcoholism may influence the decision. If with the syphilitic history there are scars in the throat, nodes on the bones, or other signs of syphilis, the conclusion will be modified by these findings. Severe pain is more prominent in syphilis than in cirrhosis, and the nodules of syphilis are markedly in contrast with the granular surface of cirrhosis.

Abscess of the Liver.

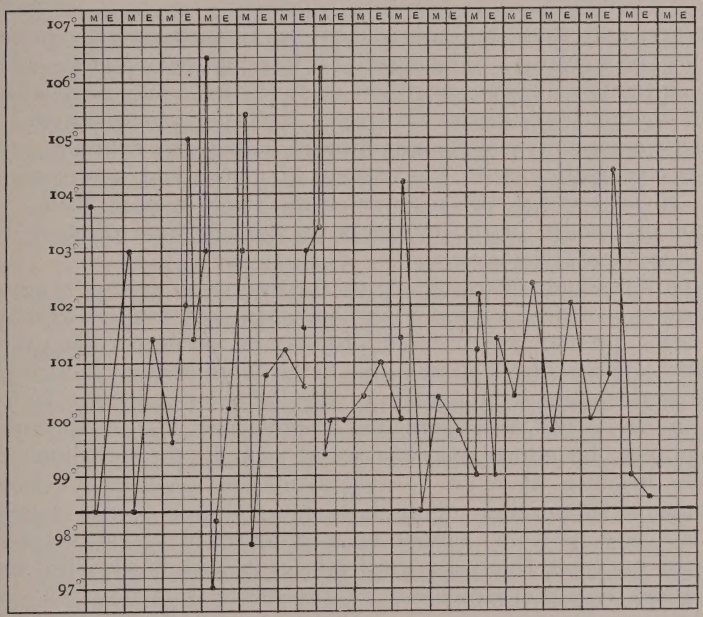
Two forms are seen: tropical abscess, so called, in which one or two abscesses are found; and multiple abscesses, found throughout the liver structure. The single or solitary abscess usually occurs in the course of dysentery, and in all probability in the amœbic form only. A single abscess may also be due to traumatism, particularly in children. Multiple abscesses occur secondarily to inflammation somewhere in the portal area. Inflammation and abscess about the rectum, inflammation of the appendix, ulceration anywhere in the gastro-intestinal tract may be followed by multiple hepatic abscesses. The abscesses, however, do not occur directly by means of emboli, as in the case of amœbic abscess, but after inflammation of the portal vein or *suppurative pylephlebitis*. Multiple abscesses of the liver also follow obstruction and suppurative inflammation of the biliary passages (*suppurative cholangitis*).

Tropical abscess, or amœbic abscess varies in its clinical course. In a typical case the clinical picture is that of the general symptoms of suppuration setting in in the course of or soon after an exacerbation of amœbic dysentery, with local symptoms referred to the liver.

Symptoms. The *general symptoms* are those of intermittent fever, paroxysms of which may occur daily or only every second day, and in which chill, fever and sweat occur. The fever may be remittent or may be continuous.

The *local symptoms.* Pain in the region of the liver; this may be referred to the region of the right or the left lobe. It may be seated in the fifth or sixth interspaces anteriorly, or behind at the ninth and tenth ribs. There may be pain in the right shoulder. The pain may be paroxysmal, or it may be intense and persistent.

FIG. 89.



Intermittent fever in abscess of the liver.

Physical Examination. On examination the liver is enlarged. The enlargement may be uniform; if the abscess is central the entire organ takes part in the swelling; on the other hand it may be an enlargement upward in the anterior, the axillary, or the posterior region. If the convex surface of the right lobe of the liver is affected the enlargement is usually upward. If the lower portion of the right lobe is affected, enlargement extends downward, and the lobe of the liver can readily be detected on palpation. The mass may extend outward from the liver edge. At first it is hard and indurated; ultimately it softens and may fluctuate. If the abscess is limited to the left lobe of the liver, and is situated about the suspensory ligament, the enlargement may be seen below the xiphoid cartilage. It may extend to the umbilicus and project forward to a great degree. Sometimes it may be so large as to cause eversion of the ribs of each side, and the entire epi-

gastrium be unusually prominent. The surface may become reddened. Over the tumor there is tenderness on palpation, and there may be, as in other situations, fluctuation. Œdema of the surface is frequently seen.

The irregular enlargement above mentioned is made out by percussion. The enlargement may be difficult to ascertain on account of secondary pleural effusion, or secondary pleural inflammation with the development of a hepato-pulmonary fistula, on account of which dulness is created posteriorly. If the case has been seen from the first, a friction sound may precede the development of the pleural complication, and the physical signs of effusion gradually develop while under observation.

The patient complains of weight and fulness in the region of the liver; the enlargement causes some dyspnœa; cough is of frequent occurrence, and from the enlargement or from the septic symptoms there may be vomiting. The appetite is lost, and nausea at the sight of food is prominent. The condition of the bowels may vary with the state of the intestinal tract at the time of the hepatic complication. The dysenteric symptoms may subside entirely or they may continue. Often there is constipation, with the passage of mucus and hardened fæces only. In an obscure case a study of the stools may be made. The detection of amœbæ in the mucus or in the intestinal discharge may point to the true conclusion.

Atypical cases are characterized by the absence of general symptoms, or the absence of local signs. Fever may be absent entirely, exhaustion alone being present, which could properly be ascribed to the previous dysentery. Pronounced anæmia due to the dysentery may be associated, as well as rheumatic inflammation of the joints, or neuritis. In a case under my care, with the exception of anæmia and loss of appetite the only symptom for a long time was severe pain in the sixth interspace. In other instances there are no liver symptoms whatsoever. General symptoms of suppuration or an irregular fever, or even a continued fever the cause of which cannot be ascertained, may alone be present. In one of my cases there was moderate continued fever, with loss of appetite and dyspeptic symptoms. There was no diarrhœa. No cause could be given for the fever, although it was noted that there was slight enlargement of the liver. The patient slipped out of the ward and went down to the yard to smoke; on his return he was seized with an intestinal hemorrhage which could not be checked and which resulted fatally. At the autopsy a large abscess of the liver was found, and there was ulceration of the rectum from which the intestinal hemorrhage took place.

The diagnosis is not difficult usually in the typical cases. Under all circumstances attention must be paid to the facts bearing upon etiology and the association of general and local symptoms. If the general symptoms of suppuration are present abscess may be mistaken for an intermittent fever. The results of an examination of the blood and of treatment by quinine would establish a diagnosis of the malarial fever. It is difficult sometimes to determine whether the abscess is in the abdominal wall or in the liver proper, or whether it is situated beneath the diaphragm. If the liver is movable with respiration, the two other conditions may be excluded. An abscess in the abdominal wall is not

influenced by respiration, and in sub-diaphragmatic abscess the movement is impaired. Suppuration of an hydatid cyst cannot be distinguished unless it has been known beforehand that a simple hydatid was present in the liver. Under such circumstances if suppuration arises, the probability of its being confined to the cyst is very great. Abscess of the liver must be distinguished from gall-stones, which are attended by intermitting fever without suppuration. While the distinction is difficult in many cases, yet the history of the case, the association of jaundice which deepens after each paroxysm, and the good general nutrition of the patient point to the latter. Abscess of the liver is of shorter duration than the former, and the primary cause of it can usually be ascertained upon examination of the rectum or upon the determination of suppuration in other parts of the body.

Exploratory puncture must be resorted to in many cases, and usually can be done with safety. Puncture must be made over the region in which the enlargement is greatest, or at which the swelling is most prominent. In abscess secondary to dysentery, a brownish-colored pus will be withdrawn resembling anchovy sauce. It may be of a peculiar odor, and on examination amœbæ common to this form of dysentery may be found. If there is no point of election, the needle may be entered in the lowest interspace in the anterior axillary or the seventh interspace in the mid-axillary line. A fairly large sized aspirator should be used. Suppuration may be present, and yet not be reached by aspiration.

The complexion in tropical abscess of the liver is peculiar, and has been insisted upon by all writers upon tropical diseases. The skin is sallow, the complexion muddy, the face pale. Through this a slightly icteroid tint may be seen, and the conjunctivæ are bile-tinged. Jaundice is of rare occurrence.

Abscess of the liver may also be due to pyæmia. It may be a part of general pyæmia or, as previously mentioned, of portal pyæmia. Parasites and foreign bodies, as well as gall-stones, may excite an abscess.

The echinococcus cyst may suppurate, or round-worms may penetrate to the liver and cause suppuration.

The symptoms of *suppurative pylephlebitis* and of *pyœmic abscess* are general and local. Jaundice is more common than in solitary abscess, and there is greater pain and tenderness over the liver, which is uniformly enlarged and tender. With the enlargement of the liver and jaundice we have the symptoms of pyæmia. They are not peculiar. Sometimes the fever is distinctly intermitting, or it may be irregular and septic in character.

The symptoms of solitary abscess of the liver, as has been previously stated, may be obscure, and attention be called to the liver only when symptoms due to a rupture in the neighboring organs may arise. If perforation takes place into the peritoneum it is not likely that the cause can be established during life. A frequent direction in which the extension takes place is through the diaphragm to the pleura, then to the lung. An empyema may be set up, the true source of which may not be ascertained unless an examination of the pus is made. The physical signs are those of empyema—dulness or diminished resonance, absence

of fremitus and vocal resonance, diminished breath-sounds, and lessened movement, with the occurrence of symptoms of cough and dyspnœa. When the lung is infected the physical signs may resemble those of consolidation. There are dulness, bronchial breathing, and increased tactile fremitus. A harassing convulsive cough occurs, and sooner or later expectoration of a reddish-brown, brickdust-colored material which resembles anchovy sauce. This characteristic expectoration is decisive. Amœbæ are found, and, in addition to blood pigment and corpuscles, orange-red crystals of hæmatoidin, cholesterin plates, and leucin and tyrosin may be seen. When the abscess perforates into the stomach or bowel the discharge from either cavity may be of the above-mentioned nature. Perforation into the pericardium is followed usually by immediate death.

Hydatid Disease of the Liver.

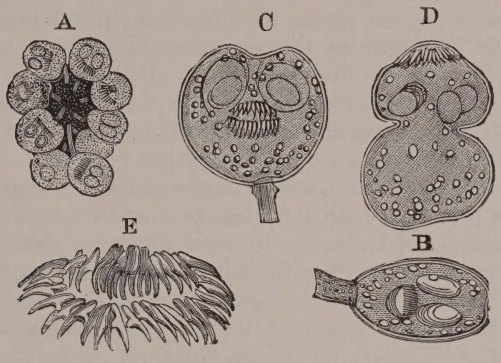
Hydatid disease is comparatively rare in this country, and yet, without doubt, at least from my own experience, is increasing in frequency. Without an increase in the opportunities for observation, I have seen seven cases within the last two years, compared to the same number during the five previous years. The disease occurs in people who are associated with dogs. It may occur at any age, but is most common in adult life. It is very rare before the fifth year. The symptoms are local, depending upon the size of the tumor. Small cysts may be present without any disturbance. Large and growing cysts cause signs of tumor, with great increase in the size of the liver. The physical signs depend upon the situation of the tumor. It may be found in the median line above the umbilicus, causing a distinct prominence, tense and firm, which sometimes yields fluctuation. Quite often the tumor grows at the suspensory ligament, pushing the diaphragm upward, dislocating the heart, and causing an increased area of dulness in the left upper quadrant. In this position it may simulate a pancreatic cyst or effusion in the lesser peritoneal cavity. If the tumor is in the right lobe the enlargement of the liver may be upward or downward. The upper border of liver dulness may begin two or three interspaces higher than normal posteriorly or in the axillary region. If the cysts are superficial when palpated with the fingers of the left hand and percussed with the right, a vibration or trembling movement is felt, which may continue for a certain time. It is known as the *hydatid fremitus*. It is not always present. The enlargement is painless. Local sensations of weight and dragging may be complained of. If suppuration sets in there may be considerable pain.

The general symptoms are negative; the nutrition does not suffer unless the enlarged mass interferes with physiological acts of digestion and assimilation by pressure. If suppuration sets in the general symptoms of abscess of the liver arise. Jaundice is more common than in tropical abscess. The abscess may perforate into one of the adjacent hollow viscera, or into the pleura and bronchi. It may perforate externally. It may perforate into the pericardium or vena cava, and cause death. If perforation takes place in the biliary passages obstructive jaundice

arises, with secondary suppurative cholangitis. When the cysts rupture, or if they are aspirated, an eruption of urticaria may break out. This is not of diagnostic significance, except that it may point to rupture of the cyst.

Diagnosis. The diagnosis is not difficult. The occurrence of irregular, painless enlargement of the liver without general symptoms is significant. If fluctuation is detected, or the fremitus, a more positive conclusion can be reached. When suppuration takes place the symptoms

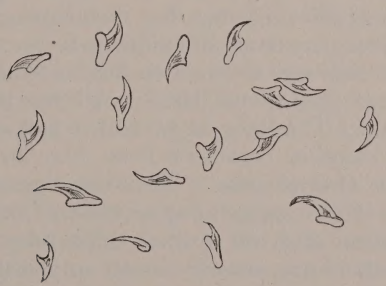
FIG. 90.



Human echinococci. (From FINLAYSON, after DAVAINE.)

A, a group of echinococci, still adhering to the germinal membrane by their pedicles. × 40.
B, an echinococcus with head invaginated in the body. × 107.
C, the same compressed, showing the suckers and hooks of the retracted head.
D, echinococcus with head protruded.
E, crown of hooks, showing the two circles. × 350.

FIG. 91.



Hooks from tænia echinococcus. × 350.

are like those of abscess of the liver. Hydatid disease must be distinguished from *syphilitic hepatitis*, in which there is irregular enlargement. The enlargement is hard and does not fluctuate. Sometimes the symptoms resemble cancer, but the age of the patient, the occurrence of jaundice, the extreme emaciation and cachexia look to that affection rather than to hydatid disease. *Enlargement of the gall-bladder* containing a mucoid fluid, in which fluctuation can be detected, may simulate hydatid disease. The enlargement, however, may be preceded by con-

ditions which cause obstruction of the cystic duct. The gall-bladder is movable. In some instances there may be resonance between it and the liver. It is of a pyriform or oblong shape usually. In *hydronephrosis* the symptoms of a localized cyst are present. It does not move with respiration, as in hydatid disease; is attended by symptoms of renal disease; the results of exploratory puncture must occasionally be awaited before a diagnosis can be established. A hydatid cyst may frequently be confounded with *pleural effusion* of the right side. The physical signs of effusion at the right base may be present. The distinction may be made by the character of the line of dulness. In hydatid cyst, as Frerichs points out, it is a curved line, the greatest height of which is found in the scapular region. It is not difficult usually to distinguish hydatid cyst from other forms of painless enlargement. In *fatty* and *amyloid* disease the enlargement is uniform. Both are of common occurrence in individuals of previous ill health, whereas hydatid disease occurs in healthy individuals.

An absolute diagnosis of hydatid disease is formed upon the results of exploratory puncture. When this is made over a tumor, or the centre of dulness, if it is due to hydatid disease a clear fluid, slightly opalescent, is withdrawn. The fluid is of a specific gravity of 1005 to 1018; it is of neutral reaction, does not contain albumin, but contains chlorides and sometimes traces of sugar. Hooklets may be found in the clear fluid.

Diseases of the Gall-ducts.

Catarrhal Jaundice. This is due to inflammation and obstruction of the terminal portions of the common bile-duct. But few words need be said, as it has been referred to frequently in speaking of jaundice. The symptoms are those of jaundice in moderate degree, occurring coincidentally with or following in a few days upon an attack of acute gastritis. The disease may occur in epidemic form. The onset is more severe, attended by chill and fever, with headache and vomiting. The temperature does not go beyond 102°. All the signs of obstructive jaundice are present. The liver is normal in size or slightly enlarged and tender. The jaundice continues from four to eight weeks, but may disappear in a shorter time. The first sign of the relief to the symptoms is shown in change in the appearance of the stools. The clay-colored stools disappear and the normal color returns. The affection, especially the epidemic form, usually occurs in young subjects.

The *diagnosis* is based upon the age, the association of the jaundice with gastritis, for which frequently a definite cause can be ascertained, and the absence of organic heart disease, or any lesion within the body, on account of which jaundice might arise; the moderate degree of jaundice, the absence of emaciation and symptoms of portal obstruction, the occurrence of moderate enlargement without pain. It must not be forgotten that jaundice due to obstruction from gall-stones, or to pressure from tumors outside of the duct, is characterized in its onset by phenomena similar to those just mentioned. It is often necessary to wait before an opinion can be given, although a history of the previous attacks of

jaundice and the age of the patient, after forty, also lead to caution in the diagnosis.

Gall-stones. Gall-stones form in the biliary passages and may remain therein without creating symptoms, or in the effort at passage cause attacks of pain called *hepatic* or *biliary colic*, after which the stone may pass into the intestinal tract without further hepatic symptoms. It may become obstructed in the biliary canal and set up catarrhal or suppurative inflammation, which in turn is followed by stricture in many cases. Gall-stones usually form or at least show signs of their presence in patients after forty, most frequently in women and in people who have led a sedentary life and partaken of rich and indigestible food. Individuals in different generations of the same family are predisposed to them.

Hepatic Colic. The passage of a gall-stone may be attended by a slight amount of pain only, so that if not in the right upper quadrant it would pass for an attack of simple indigestion. In the large majority of cases the pain is severe. The attack may be preceded by biliousness or indigestion for twenty-four hours, and moderate pains or a sense of weight and fulness in the liver. It frequently follows the taking of food. Ringing in the ears, disturbance of vision, or undue flushings are said to precede it in some instances.

The attack may be sudden. The patient is seized with pain which is usually complained of along the margin of the ribs at the right border, or there may be pain above the ribs over the liver, and in the right shoulder at the same time. From the hepatic region it extends to the median line. It may be most pronounced in this locality from the first. The pain is intense and paroxysmal. The patient is doubled up in agony. It causes more or less collapse. The pulse increases. Vomiting usually occurs at the same time, first of the contents of the stomach, then a yellowish bile-stained fluid. The vomiting may be extreme, so that the patient is tormented by the pain, the retching, and vomiting. The attack sometimes disappears as suddenly as it occurred, or wears off gradually. When most severe the symptoms of shock follow. The bowels are not disturbed during the attack. The urine may become suppressed; it is high-colored. After the attack it may contain bile.

At the time of the attack there is considerable tenderness below the xiphoid cartilage and in the hepatic region. The tenderness is more marked when deep pressure is made in the gall-bladder region and to the right of the mid-clavicular line, at the margin of the ribs. The epigastrium may be slightly swollen. The tenderness persists after the attack, and the stomach may be weak and irritable for some time; pain, however, is usually removed at once. The attack may frequently recur until the stone has been passed, so that in twenty-four hours the patient may have a dozen or more attacks. When the attacks have subsided light jaundice may supervene, which usually does not continue more than a week at the furthest, during which there are also symptoms of mild gastritis.

In some instances a chill precedes the pain, or immediately follows it, after which the temperature rises. When the paroxysm subsides the

fever disappears rapidly, being followed by profuse perspiration. If the gall-stones have set up catarrhal inflammation moderate fever may continue for a few days.

During the paroxysms of hepatic colic a gall-stone may be passed. It is desirable to determine this if possible. This can only be done by careful examination of the fæces by placing them in a sieve and pouring water upon them until they become soluble. Instead of gall-stones, dark-colored granular bile, which has become inspissated, is sometimes seen in the motions. This character of bile gives rise to as much pain, according to Harley, as true biliary concretions. If the stone is not passed it may fall back into the gall-bladder and not cause further symptoms for a time, or become impacted in the ducts. The impaction may be such that obstruction is not caused by its position, the bile being forced through or around it, or complete obstruction may take place. (See Jaundice.)

Obstruction of the Ducts. The symptoms from obstruction depend upon its seat. If the obstruction is in the *cystic duct* the gall-bladder enlarges. The liver is not secondarily affected. The enlargement is noted at the edge of the liver in the usual situation, and may gradually increase to an enormous extent, so that it has been mistaken for an ovarian cyst. The gall-bladder is often quite movable, and on account of its location and movability, as well as its long shape, has been taken for a floating or movable kidney. If not too large, when the hand is placed over it and along the margin of the liver on careful deep palpation, if the patient takes a full breath, the rounded or pyriform mass can be felt to swell underneath the palpating fingers. The enlargement is not attended by other symptoms except mechanical, unless the contents of the gall-bladder are purulent. In obstruction with simple enlargement the fluid of the gall-bladder, should aspiration be performed, is thin, of a mucoid nature, alkaline in reaction. It may contain cholesterin plates, and sometimes blood. It must be distinguished from the fluid of an hydatid cyst.

Acute phlegmonous inflammation of the gall-bladder may take place attended by localized pain and tenderness, by high temperature, extreme prostration, and the rapid development of the typhoid state. Peritonitis rapidly ensues. It could not be distinguished from other forms of acute inflammation in the same region, unless there was (1) a history of gall-stones; (2) tumor of the gall-bladder before the attack developed. *Suppurative inflammation* of the gall-bladder may occur The enlargement may increase, the tumor becoming tender and painful on palpation. The direction of growth is toward the umbilicus. The general symptoms are those of suppuration. Hectic fever or markedly remittent fever occurs, and unless surgical relief is given peritonitis from infection or from rupture takes place. This may be suspected by the occurrence of collapse and increase in extent of the local symptoms.

Tumors of the *gall-bladder*, usually due to cystic obstruction, as previously mentioned, may be mistaken for floating kidney, for tumor of the pylorus, and for ovarian cyst.

Tumors of the gall-bladder from either of the above-mentioned causes are recognized by their position and shape, and by the character

of the tumor. The *position* varies. The usual site is in the gall-bladder region, but it may extend as low as the groin, or may be so large as to distend the ribs and fill almost the entire abdominal cavity. If the case, however, has been under observation the tumor originally would have been found upon search in the gall-bladder region, the location of which is fairly definitely settled: this is the point corresponding to the bisection of the border of the ribs by a line drawn from the acromion process of the right shoulder to the umbilicus. From this point the tumor grows toward the umbilicus in nearly all the cases. The shape is pyriform or globular, and can be recognized by this shape. The *character* of the tumor varies. It is usually tender, firm, but elastic on pressure and movable. Fluctuation may often be detected. If the enlarged gall-bladder contains calculi they may be felt as small, hard masses which cause a grating sensation to be transmitted to the finger. On aspiration, if the cystic duct is obstructed, the mucoid fluid previously mentioned, or pus, is withdrawn. If the common duct is obstructed bile would pass through the trocar.

The enlargement must be distinguished from tumors of the liver, of the stomach, duodenum, pancreas, or lymphatic glands. Tumors of the liver are usually due to *carcinoma*. They are multiple, associated with enlargement of the liver, with jaundice, ascites, enlargement of the spleen, and emaciation. Tumors of the *stomach*, *duodenum*, and *pancreas* are in a different position, and are attended by functional disturbance of the respective organs from which they spring. An *abscess* of the liver may simulate enlargement of the gall-bladder if purulent. If the abscess can be palpated an area of induration is first felt, followed afterward by softening and fluctuation of the swelling. In judging of the true nature of the tumor consideration of the causes of abscess must be made. In *hydatid* disease the tumor develops slowly; it is painless; it may yield fremitus, and if movable the course is slow and not attended by general symptoms. *Multilocular hydatid disease* can rarely be distinguished save in the difference of the position of the tumor. It is nodulated, hard, and tender, but is associated with jaundice, ascites, œdema of the legs, enlarged spleen, and great emaciation and prostration, with rapid decline of the patient. A *syphilitic gumma* in the liver may occupy the region of the gall-bladder. It can usually be made out as continuous with the liver structure. It is tender and painful, but irregular; other signs of syphilis, or a history of the infection and of symptoms of a primary and secondary period will aid in the distinction of the disease.

Floating kidney. The gall-bladder is larger and more movable at one end than the other; whereas the entire kidney is movable. The gall-bladder may fluctuate and the mass is associated with symptoms of hepatic disease. On the other hand, the well-known symptoms of floating kidney, the shape of the tumor, the sensation of nausea secured by palpation, point to the renal origin of the mass. Tumors of the kidney must be distinguished, such as sarcoma, hydronephrosis, and pyonephrosis. 1. There may be changes in the urine. 2. In renal tumors the intestine is in front of some portion of them, or a zone of resonance is found between the liver dulness and the tumor. 3.

Renal tumors are fixed. They may, as in hydronephrosis, come and go, preceded by attacks of renal colic and attended by *anuria*. From *ovarian* or *uterine tumors* the diagnosis must be made by examination of the genital organs.

Enlargement of the gall-bladder on account of calculous obstruction must be distinguished from enlargement due to *cancer* of that organ. This is often difficult and cannot be done without having the patient under observation for a long period of time. Cancer of the gall-bladder is usually primary. It may begin in the gall-ducts. In the larger number of cases it occurs in patients who have had gall-stones. It is found most frequently in females, and after the fiftieth year. Tight lacing or pressure around the abdomen may predispose to it. The symptoms are those of pain, jaundice, emaciation, cachexia, and the presence of a tumor. The pain is localized and lancinating in character. Jaundice occurs in 70 per cent. of the cases, and gradually increases in intensity. The tumor is situated in the gall-bladder region, to the right of the umbilicus. It is hard or firm, painful, and the seat of tenderness. The tumor is fixed. Sometimes the disease is found in the cystic duct, and then the gall-bladder is enlarged. As the history of gall-stones is of frequent occurrence in both instances, it is impossible to distinguish the two forms of obstruction causing enlargement, save that in carcinoma the emaciation and cachexia may point to the true nature of the case. In tumor of the gall-bladder due to cancer, the secondary effects on the liver are usually more marked than in tumor from other causes. The liver enlarges and its surface becomes irregular or nodular.

Obstruction of the common duct by gall-stones. (*a*) In addition to jaundice, paroxysms of chill, fever, and sweat occur, with catarrhal inflammation of the biliary passages. (1) The paroxysms resemble intermittent fever; (2) the jaundice may continue for years and deepen after each paroxysm; (3) with the paroxysm, hepatic colic may occur; (4) health fails but slightly. The paroxysms may occur daily, or only once a week, or they may be tertian and quartan in type. On account of the obstruction in this situation the liver becomes enlarged. It is firm and smooth on palpation. The enlargement, as determined by percussion, is uniform. (*b*) Gall-stones may cause suppurative inflammation of the biliary ducts, just as suppuration of the gall-bladder may ensue. The symptoms, both general and local, are pronounced. The fever may be intermittent, but is more likely remittent; jaundice is present, but constant in its intensity. The local signs of enlargement and tenderness are made out. The patients die of exhaustion or septicæmia. Sometimes the gall-bladder ruptures into the stomach or colon, and temporary abeyance of the symptoms may result.

The Accidents of Gall-stones. While the effects just noted of the presence of stones in the biliary passages may rightly be considered as accidents, nevertheless their occurrence is so common as to be part and parcel of the history of gall-stones. As accidents, we have the formation of biliary fistula, with passage of the gall-stone into the contiguous organs or channels. The stone may ulcerate into the gall-bladder from one of the ducts, may perforate the portal vein, may perforate into the abdominal cavity—the most frequent accident. Perforation is

of common occurrence also into the duodenum, into the colon, and rarely into the stomach. The occurrence of such perforation can only be assumed by its secondary effects: (1) An attack of gall-stones; (2) local inflammation with fever; (3) the occurrence of peritonitis, or the discharge of pus by the bowels, or by vomiting. That it is due to gall-stones is proven in those rare instances in which the stone is passed per rectum. Often it may be impacted in the intestinal canal, causing the symptoms of acute obstruction, or in the rectum, causing local tormina and tenesmus. The perforation, however, occurs in other directions. Sometimes fistulous connection is formed between the gall-bladder and the urinary passages, calculi and pus being discharged from the urine. In other instances fistulæ between the bile passages and the lungs take place. The bile is coughed up and expectorated sometimes with small calculi. The most common form is for the ulceration to take place toward the surface with the formation of cutaneous fistula. After the fistula has opened externally gall-stones in large numbers may be passed. If not, the diagnosis of the cause of the fistula must be based upon the history and the results of investigation by probe, with attention to the condition of the other organs.

Symptoms. In stenosis of the bile-ducts the chief symptom is that of jaundice. Colicky pains occurring in paroxysms, intermittent jaundice varying in intensity, and an intermittent fever, point to gall-stones. If the obstruction is due to disease outside of the ducts its nature must be inferred by the symptoms and physical signs of disease in neighboring structures. If the jaundice is due to enlargement of the lymphatic glands its nature may be inferred by determining the presence of primary carcinoma in other organs of the body, or by the condition of the lymphatic glands in other parts. If they are the seat of malignant disease this usually can be recognized. In the case of Hodgkin's disease the examination of the blood may be of service in the diagnosis. Cancer of the liver must be excluded by its symptoms—enlargement with jaundice, with moderate fever, rapid emaciation, and short duration of the disease.

Diseases of the Spleen.

Palpation and Percussion of the Spleen. The spleen lies in the left upper quadrant beneath and in contact above with the diaphragm and below with the tail of the pancreas, cardiac end of the stomach, and supra-renal capsule. It extends transversely between the upper border of the ninth rib and the lower border of the eleventh rib, and from the middle axillary line posteriorly toward the spine.

An enlarged spleen usually retains the shape of the normal organ. It is accessible to *palpation* in proportion to the degree of the enlargement of the organ, and of relaxation of the abdominal walls. When moderately enlarged the smooth, blunt, rounded anterior surface and sharp edge of the spleen can be felt at the margin of the ribs in deep inspiration; and when the enlargement is great, as in *leukæmia*, the organ can be grasped with both hands, and its hilus clearly mapped out. The same thing can be done in the rare instances of *floating*

spleen, but here a knee-chest position will favor successful palpation. In splenic leukæmia the spleen may be larger after a meal, yield a creaking fremitus on palpation, a murmur on auscultation, and may even pulsate. The spleen may also lessen in size following diarrhœa or free hemorrhage. As it lies entirely behind the ribs when of normal size, of course it does not admit of palpation.

PERCUSSION. Being a solid body it gives a dull sound on percussion, contrasting with pulmonary resonance above, intestinal tympany below, and stomach tympany anteriorly. Posteriorly and below its dulness merges into that of the lumbar region and kidney. The upper posterior portion is hidden behind the diaphragm and overlapping lung, and hence is not accessible to percussion. Practically, therefore, the normal splenic dulness extends between the ninth and eleventh ribs, in the middle axillary and posterior axillary lines, the spleen being there in contact with the ribs.

In percussing the spleen the patient should lie on his right side. Beginning from above downward we percuss gently until pulmonary resonance is succeeded by dulness; and then anteriorly, proceeding toward the axilla, until stomach tympany yields to dulness. In the same way, percussing from below upward, the line is reached where intestinal tympany gives way to dulness.

The spleen may be compressed by a stomach or colon distended with gas, and its dulness may appear increased through distention of the stomach and colon with solid matter, or by a left pleural effusion, or left basal pneumonia. The spleen may also be pressed up by ascites or by a large abdominal tumor, so that its normal dulness is much lessened.

If the ligament which holds it in place become relaxed, the spleen may become floating. According to Stintzing a floating spleen is increased in density, is generally enlarged, and is recognized by its form (notch, etc.), by being movable to and fro, and by the absence of splenic dulness in its normal position and its reappearance when the spleen is replaced.

Enlargement of the spleen may be acute or chronic. *Acute enlargement* occurs in certain infectious diseases, particularly typhoid fever, typhus, smallpox, relapsing fever, scarlet fever, diphtheria, epidemic cerebro-spinal meningitis, the malarial fevers and meningitis, in diseases with blood poisoning, as septicæmia, puerperal fever, and erysipelas.

A rare cause of enlargement is *acute splenitis*. Generally it is the result of emboli lodged in it and starting from an endocarditis. The area of splenic dulness is increased rapidly, and there are local pain and tenderness on pressure, increased by coughing and deep inspiration; other symptoms are fever, nausea and vomiting, and occasionally delirium. If, as frequently happens in splenitis, emboli lodge in the kidneys also, the urine will be albuminous and bloody. If suppuration ensue the fever becomes hectic and the spleen continues to increase in size. *Splenic abscess* may, however, remain latent until rupture occurs.

Enlargement of the spleen can be distinguished from enlargement of the left kidney by the greater movability of the spleen. 1. The

spleen does not extend as far back toward the spine as the kidney, so that the fingers can be thrust behind its posterior border, and if the other hand grasp the anterior edge the organ can be moved backward and forward. Splenic dulness extends to the ninth rib or higher. Kidney dulness has no thoracic area, but reaches to the spine (lumbar). 2. Again, the spleen is movable with respiration, while the kidney is not. 3. The spleen falls farther toward the median line, when the patient is in the knee-chest position, than the kidney does. 4. An enlarged kidney has the colon in front of it, and hence its dulness is obscured by the tympany of the bowel. 5. The shape of an enlarged kidney is more globular than that of the spleen. The anterior surface of the latter is smooth and rounded, but at its junction with the flat posterior surface there is a sharp edge. 6. Pain in renal diseases often shoots down the ureters and into the testicles. In diseases of the spleen the pain is generally localized to the splenic region, and may shoot into the left shoulder. 7. Result of examination of the urine will often make clear that the disease is renal, or, by its negative result, will point to the spleen as the cause of the tumor.

Chronic enlargement of the spleen occurs as hypertrophy and as the result of amyloid disease, leukæmia and pseudo-leukæmia, chronic malarial poisoning (ague-cake), syphilis, hydatid tumor, and cancer. Enlargement is greatest in leukæmia and in ague-cake. The spleen in well-marked cases of these affections may reach to the umbilicus and even beyond, filling up the hypogastrium and extending to the right iliac region, measuring thirteen or fourteen inches in length and half as much in breadth, and proportionately increased in thickness.

Diagnosis of Enlargement of the Spleen. The *diagnosis* of *splenic leukæmia* rests principally upon the blood condition, particularly upon the existence of a marked increase of white blood-cells. Red cells are decreased, and altered forms are present. In addition to characteristic blood changes there is a great disposition to hemorrhages; dropsies and priapism are common; and in the late stages fever, diarrhœa, great weakness, and grave complications, such as pneumonia, occur.

Hemorrhage in splenic leukæmia occurs from the nose, bowel, stomach, gums, or kidney. It may also be subcutaneous, intermuscular, cerebral, or retinal.

Regarding the diagnosis of *splenic hypertrophy* (ague-cake) in *chronic malarial affections*, Osler says: "The history of malarial cachexia, the absence of lymphatic enlargement, and the blood condition, will usually be sufficient for the purposes of a diagnosis. Great increase in the white blood-corpuscles is not often seen in the chronic splenic tumor of malaria; indeed, they may be much diminished in number. Toward the end in very chronic cases the clinical picture may be very similar; the large abdomen, possibly ascites, dropsy of the feet, and irregular fever may resemble closely splenic leukæmia, and the absence of an increase in the colorless corpuscles may be the only marked difference."

Amyloid spleen with enlargement of the organ occurs in conditions attended by prolonged suppuration, especially when the bones are involved, and in chronic phthisis and syphilis. The spleen is en-

larged, hard, and painless. The enlargement is rarely great enough to produce distress on that account, and it is so commonly associated with a similar condition of the liver and kidneys, if not of other organs, that any constitutional symptoms produced by the spleen are apt to be masked by those produced by other organs.

Hydatid tumor of the spleen rarely causes any symptoms except when it becomes very large; then it may give rise to discomfort and a dragging pain in the left hypochondrium. But hydatid tumors of the spleen are only exceptionally very large; when large enough to admit of palpation, and when the tumor is situated anteriorly or projects from the lower border or from beneath the organ, the detection of fluctuation, the withdrawal of the characteristic cystic fluid by aspiration, and possibly the hydatid fremitus, will establish the diagnosis, when taken in connection with the gradual development of the tumor and exposure to possible infection. In the absence of physical signs of a cyst, the diagnosis can only be suspected from the habits of the patient or his place of residence. Suppuration of the sac may be brought about by injury, or rupture into the adjacent cavities with grave, if not fatal results.

Inherited syphilis and *chronic syphilis of considerable duration* are accompanied by enlargement of the spleen. They cause a chronic interstitial inflammation. The enlargement is not very great, and does not possess characteristic features.

Malignant tumors of the spleen are very rarely primary. The diagnosis must be made by noting malignant disease elsewhere, the very rapid enlargement of the spleen, with possibly nodules scattered over its surface, and the presence of cachexia and the usual constitutional signs of a malignant disease.

In young children enlargement of the spleen is not uncommon. It is found associated most frequently with rickets, syphilis, and malarial poisoning, and has been attributed to each of these diseases as a cause. In the London *Lancet*, April 30, 1892, Dr. J. W. Carr analyzes thirty cases, and comes to the conclusion that the enlargement of the spleen is due to *splenic* anæmia, the essential cause being unknown. Rickets, syphilis and ague are found as passing causes only, since the disease is found in some cases where these causes can be excluded. According to this author, the disease is extremely rare in children older than two and one-half years.

Diseases of the Pancreas.

The function of the pancreas, or at least isolation of its functional activity from the functions of organs physiologically associated with it, is surrounded by so much obscurity that diseases of the pancreas are attended by the same obscurity. As the pancreatic secretion aids in intestinal digestion, particularly in emulsifying fats, symptoms due to disturbance of this function are looked for, and it is true in a measure in all cases of pancreatic disease that there is some *intestinal indigestion*. For the purpose of determining whether the function of digestion of fats has been modified, the patient with suspected pancreatic disease is given

fats in some form and the stools watched. If fat is passed in the stool in the amount taken by the mouth, without being broken up, or emulsified, it is held as proof that disease of the pancreas is present. While *fatty stools* may be indicative of pancreatic disease, the absence of fat in the stools in patients who are fed upon it cannot be used as a means of the exclusion of disease of this organ, for notwithstanding its absence in a large number of instances in which the experiment was tried, the pancreas was found to be the seat of extensive disease. Sugar has been observed in the urine in many cases in which the pancreas was the seat of the disease. In fact, *glycosuria* has been attributed to pancreatic disease in cases of grave diabetes. This symptom, however, is not constant in pancreatic lesions.

The three classes of symptoms just mentioned are, therefore, not diagnostic of pancreatic disease, but afford presumptive evidence of its presence. Most striking symptoms of disease of the pancreas, apart from that which is due to a morbid process, as suppuration or cancer, are the symptoms due to a *tumor* pressing upon surrounding structures. It may press upon the gall-duct, causing jaundice. It is one of the most frequent causes of obstructive jaundice. Finally, some diseases of the pancreas may cause a tumor situated in the epigastric region which may resemble an aneurism, a tumor of the pylorus, or of the transverse colon. Tumors of the pancreas are usually due to *cancer*. This is usually of the scirrhus variety, and generally primary. The enlargement cannot be distinctly made out unless the patient emaciates very much. When it has advanced considerably it may simulate aneurism, but is distinguished by the difference in the character of pulsation. In aneurism the pulsation is distensile, in disease of the pancreas it is an up-and-down movement. The hand is lifted with each pulsation of the aorta. Tumor of the pylorus is excluded largely because of the more superficial position of the mass, because of its association with pyloric obstruction, and with less frequent jaundice than in disease of the pancreas. A pyloric tumor is more movable and may change position after the stomach is inflated by gas or distended by fluid. Examination with the patient on the hands and knees may aid in the distinction between the two. In a tumor of the transverse colon its nearness to the surface and movability, its association with more or less constipation, with occurrence of intestinal hemorrhage, are of diagnostic significance.

The general symptoms of the cancerous cachexia; the occurrence of intestinal indigestion, or of fatty stools; the gradual onset of jaundice; epigastric pain, which is complained of as deep-seated; an immovable tumor, with glycosuria, make a symptom-group very characteristic of *cancer of the pancreas.*

Hemorrhage. We owe to F. W. Draper and Prince our knowledge of hemorrhage into the pancreas. Since their labors the affection has been frequently recognized. The attack comes on suddenly in the midst of perfect health, and usually terminates life in a short period. Nothing in the occupation or conduct of the patient at the time favors the development of the hemorrhage. He is seized with severe pain, which is localized in the upper part of the abdomen. It increases in severity, is most intense in character, and may intermit like colic.

Nausea and vomiting take place almost at the same time. The vomiting becomes obstinate. Extreme depression rapidly sets in and the patient becomes anxious and restless. Collapse ensues in a short time. The extremities become cold and the forehead is covered with sweat. The pulse increases in frequency and rapidly diminishes in strength. It soon becomes imperceptible. The pain and vomiting call attention to the upper abdomen. It is tender on pressure; this may extend throughout the entire upper half of the abdomen. Tympanites may develop. There is constipation in many of the cases. The temperature remains normal, or becomes subnormal. The pain, the vomiting, the anxious and restless state continue without relief.

From the above group of symptoms it can readily be seen that the diagnosis is obscure. It can be taken for perforation of the stomach by ulcer, although the vomiting may not be so persistent and frequent. Intestinal obstruction in the upper portion of the tract presents allied symptoms. The hemorrhagic symptoms, however, are more pronounced in pancreatic hemorrhage. Pallor of the face is sure to ensue. The vomiting is not fæcal in character. Constipation can be relieved. It is, however, difficult and in many cases it may be impossible to establish a diagnosis. The rapidity of development of the symptoms is of importance. The pain and collapse may be due to rupture of an aneurism of the aorta.

Acute Hemorrhagic Pancreatitis. To another Boston professor we owe our knowledge of this disease; at least to Fitz we are indebted for collating the facts from the literature to which are added the results of his own valuable observations, by which we can recognize this affection during life. A patient with hemorrhagic pancreatitis has been previously subject to attacks of indigestion, attended by pain and vomiting; many use alcohol to excess. The attack develops suddenly, resembling somewhat hemorrhage of the pancreas. There is violent pain which is at first complained of in the upper abdomen, although it is sometimes general. Nausea and vomiting are present in all the cases; constipation in most of them. The abdomen is frequently the seat of tympanitic distention. Collapse symptoms supervene, although fever may occur. The cases terminate before the fourth day, sometimes earlier. The pain and collapse are probably due to swelling which involves the cœliac plexus.

The symptoms resemble *intestinal obstruction*. In several instances laparotomy has been performed for the relief of supposed obstruction. The intense pain in the epigastrium, the violent vomiting and distention of the upper abdomen, without a possible cause for obstruction, are favorable to acute pancreatitis. The difficulty of diagnosis, however, is so great that resort to laparotomy is justifiable in order to determine exactly the nature of the condition.

Suppurative Pancreatitis. Fitz has found that this affection occurs in adults under forty, more frequently in males. Symptoms continue during several weeks, and may persist for a year. Pain in the epigastrium is complained of, associated with irregular vomiting, the latter persisting in spite of care as to feeding. Fever is irregular in type, and exhaustion ensues. In the case under my observation, obstruction of the

portal vein took place, with ascites. The latter was large, and recurred rapidly after tapping. In this patient the pain and gastric disturbance were absent. There was no fever. Emaciation, constipation, and a tumor above the umbilicus were present; the emaciation was extreme. The tumor was ill-defined, painless, apparently superficial. Many other symptoms of pancreatic disease pointed out by Roberts were present. Apathy and despondency were marked; bronzing of the face was also present. The patient was a middle-aged man, aged forty-two, addicted to the use of alcohol. He was thought to have cirrhosis of the liver. As happened in my case, the pus may accumulate in the duodeno-jejunal fossa and fill up the cavity of the lesser peritoneum, with more pronounced symptoms of tumor than occur in similar fluid accumulations in the above-mentioned cavity.

Gangrenous Pancreatitis. This may follow later upon hemorrhages into the pancreas. The symptoms are extremely obscure during life. Symptoms of collapse may occur, following pain, which is of longer duration than in the acute form, or vomiting, which is not so persistent. In my case a patient upward of sixty years suffering from dyspepsia vomited blood during the course of an illness which was characterized by loss of flesh and weakness. The anæmia became very profound after the gastric hemorrhage, and exhaustion was extreme. There was no marked tumor, but resistance in the region below the xiphoid. There were dulness and tubular breathing at the base of the left lung. Fever was absent. Death ensued from exhaustion. A small, flat carcinoma was found in the pyloric end of the stomach, but there was no perforation. Gangrenous pancreatitis, with signs of an ante-mortem hemorrhage, was found. The accumulation took place behind the stomach and colon, but in front of the kidney; its outer wall was bounded by the spleen. It was circumscribed above by the diaphragm. Pleuritis and small pulmonary abscesses at the base of the left lung were found.

In some instances the pancreas has sloughed into the bowel, and in two such cases recovery took place after its discharge from the rectum.

Chronic pancreatitis is not recognized during life, although its possible presence must be considered in all cases of diabetes.

Cyst of the Pancreas. Cysts of the pancreas follow impaction of calculi in the pancreatic duct; sometimes the biliary calculi obstruct the orifice. The symptoms are those of tumor in the upper abdomen, which occupies the median position, or is chiefly on the left side in the upper quadrant. It may fill the abdominal cavity and simulate ovarian tumor. It usually develops slowly, but cases of rapid onset have been described. Fatty diarrhœa is not present. There is a sense of weight and fulness in the epigastrium. The cysts are not really true cysts, but accumulations of pancreatic fluid in the lesser peritoneal cavity. The signs are those of a tumor to the left of the median line, encroaching upon the left lobe of the liver above, and extending almost to the transverse umbilical line. It is smooth, and may fluctuate; it is not hard and lobulated. On account of its presence the diaphragm may be arched so that the heart is dislocated to the left and upward; the apex is found in the third interspace. It also

causes increased dulness behind on the left side, the upper border approaching the angle of the scapula. Exploratory puncture in either instance determines the nature of the fluid and may positively determine the diagnosis. (See Examination of Cystic Fluids, page 170.)

Senn has pointed out that in cysts of the pancreas the complexion is peculiar; it is described as an unhealthy yellow, dirty, or earthy hue. This writer also considers that in the diagnosis of pancreatic cyst the history of the case, the location of the tumor, and its relation to other organs are to be considered. The disease occurs in adults, and usually follows traumatism. A blow in the epigastrium is a prominent exciting cause. In some instances it occurs after an attack of so-called biliary colic or colicky pains in the upper abdomen, with vomiting, but without jaundice, characteristic of calculus in the pancreatic ducts. The growth of the tumor is unusually rapid—a point in favor of its pancreatic origin. It may attain an enormous size, as previously mentioned.

In contrast to cancer, pain is absent. Fatty stools are absent. Previous gastro-intestinal derangement may be ascertained upon inquiry. Diabetes, in this as well as other affections of the pancreas, may be present. The cyst is always found at first in the region occupied by the pancreas, depending somewhat upon the portion of the pancreas from which it originated. It may be below the right lobe of the liver, below the xiphoid, or in the left upper quadrant. In the large majority of cases it occupies the last situation. It displaces the stomach forward and to the right, the transverse colon downward, the diaphragm and the contents of the chest upward. The cyst may be movable in respiration.

Diagnosis. It must be distinguished from cancer of the pancreas or adjacent organs, aneurism, hydatid cyst of the liver, the spleen, or the peritoneum, affections of the retro-peritoneal glands, hydronephrosis, cystic disease of the supra-renal capsule, circumscribed peritonitis with exudation, ascites, cystic disease of the ovary. Pain is an important symptom of the disease of the pancreas in its more acute manifestations; it must be distinguished from the pain of intestinal obstruction and the pain of perforative peritonitis. The pain is always localized to the region below the xiphoid, or if general is confined to the upper half of the abdomen. It exactly simulates the pain of the affection just described. This is more pronounced because of the association of vomiting and collapse in this class of cases. Pain that is not so intense, of a colicky nature, attended by a diarrhœa, or constipation, in some instances with intestinal hemorrhage, may be due to *calculous disease* of the pancreas. Frequently this form of pain can be recognized if other symptoms of pancreatic disease, such as glycosuria, steatorrhœa, and intestinal indigestion are present.

CHAPTER VII.

DISEASES OF THE KIDNEYS.

The kidneys are affected by disease through several sources. First, the great vascular supply is subject to the alteration which takes place in any large arterial area either from direct hyperæmia, through the influence of the vasomotor nerves (see Hyperæmia), or through the central organ of the circulation, whereby passive hyperæmia or congestion occurs. Second, by means of the bloodvessels, thrombosis and embolism occur, particularly the latter, causing renal infarction. Third, infectious material is carried to the kidney, and in passing through the structure gives rise to the inflammations we see in infectious disease, either of an infective or simply of an irritative character. Fourth, through the means of the bloodvessels also, and by virtue of its function, the renal structure is particularly liable to irritant inflammation, for through it pass poisons that are ingested; and the products of metamorphosis which, if modified in character or increased in amount, excite irritation and lead to inflammatory changes.

But the kidney is open to attack from sources lower down in the urinary tract. Through the bladder and ureter infection may extend upward, causing the consecutive inflammatory processes, which are often seen after disease of the urethra, bladder, or ureter. It is obvious that, if changes in the urine are found, one of these three causal conditions may be present. The kidney is at the apex of a system of tubes or channels. Any alteration of them, whether mechanical or functional, has a secondary effect upon the kidney. Obstructions of the ureter or obstruction in the conduits beyond lead to consecutive hypertrophy, inflammation, and atrophy. (See Morbid Processes.)

The morbid processes which may take place in the kidney are such as are common to all organs—congestion, degeneration, inflammation, and morbid growth. The symptoms that attend the morbid processes are such as accompany similar processes elsewhere. The general symptoms of the morbid processes are not pronounced except in the case of intense inflammation with suppuration, or of morbid growths, as carcinoma, because of the small size of the kidney. We have general symptoms, on account of the morbid process, that may point to suppuration, or general symptoms due to the cancerous cachexia. Otherwise, general symptoms in renal disease are of small moment, except, as is usually the case, where there is interference with the function of the kidney. The local symptoms are only due to the morbid process, as pain in carcinoma.

The symptoms of renal disease are the symptoms of the morbid process and the symptoms due to functional or anatomical alteration of the kidney. But the structure is so closely interwoven with the function that morbid changes in one imply morbid changes in the other. As the

anatomical alterations are usually beyond the pale of investigation, again, we find functional symptoms alone are appreciated. Hence in each morbid process we look for *changes in the urine*, which is the product of renal function, and for symptoms resulting from abeyance or cessation of the function. Rarely we have *enlargements* due to tumor, as cancer or abscess, or to obstruction of the channels causing hydronephrosis, or to parasitic disease.

The urine is not alone an index of the condition of the kidneys. It varies, within the bounds of health, in color, quantity, and quality. Food, exercise, and other conditions modify the secretion. It can readily be seen, therefore, that any general disease and many local diseases cause alterations in the character of the urine. Any abnormal urine, therefore, is symptomatic of renal disease or of disease beyond the point at which the urine passes out of the body. Usually abnormal changes in the urine due to the general condition do not give rise to local renal symptoms or to abnormal renal function. The exception is seen when an excess of uric acid and urates and of oxalates is passed. They may give rise to local pain and may set up sufficient irritation to cause nephritis.

A. The general phenomena of the morbid processes are fever and emaciation. *Fever* occurs in acute nephritis, perinephritic abscess, suppurative and tuberculous nephritis, pyelitis, and with twists of the ureter in floating kidney. *Emaciation* occurs in chronic, suppurative, and tuberculous nephritis and carcinoma. The local phenomena of morbid processes are pain and tumor.

B. The symptoms due to alteration of function are: 1. *Uræmia*. 2. *Cardio-vascular symptoms*. 3. *Anæmia*. 4. *Dropsy*. 5. Changes in the character of the *urine*. 6. Changes in the frequency and character of the *micturition*. The symptoms of renal disease are, therefore, both subjective and objective.

Classification. The best classification of the diseases of the kidneys is that based upon the propositions of Delafield, who, in a paper entitled "On the Diseases of the Kidneys Popularly Called 'Bright's Disease,'"[1] submitted a classification dependent upon the nature of the morbid process. The morbid processes included congestions, degeneration, and inflammations of the renal structure. In addition to these affections we must also include in the nosology of renal disease tumors (cancer, abscess, and hydronephrosis) and anomalies of growth or position (floating kidney, horseshoe kidney), affections due to invasion of the kidney by parasites, and affections due to obstruction of the tubes through which the offices of the kidney are carried on (renal calculus, hydro- and pyonephrosis).

The Data Obtained by Inquiry. The Subjective Symptoms.

The subjective symptoms are due to morbid processes within the kidney or to alterations of its function. The class of nervous symptoms which belong to uræmia are subjective in character, as are also the symptoms of movable kidney.

[1] Trans. Amer. Physicians, vol. vi., 1891, p. 124.

Pain. 1. Pain in the kidneys is referred to the loins. It is complained of as a dull aching, sometimes increased by movement, often attended by a sense of weight or pressure. This character of pain extends over the entire lumbar region and is due to disease of both kidneys, as in acute nephritis. 2. We have, further, pain referred to one kidney. The pain may be seated in the region of the kidney behind, opposite the two lower dorsal and two upper lumbar vertebral spines; or is complained of as deep-seated, in the abdomen to the right or left of the spinal column below the level of the umbilicus. Pain on one side of the back is not generally mistaken for pain due to other causes. It may arise from myalgia of one side, or be due to disease of the vertebræ. If myalgic it may be associated with pain in other muscles and follow exposure to cold. Neuralgia of the kidney no doubt occurs. It may be due to malaria, lead poisoning, gout, or anæmia. It partakes of the characters of neuralgia elsewhere.

Pain in the situations just mentioned is usually unilateral, and may be constant or paroxysmal. *Constant* pain is usually due to organic disease of the kidney, as carcinoma, or tuberculosis. It may, however, be due to the impaction of a calculus in the pelvis of the kidney. *Paroxysmal* and *lancinating* pain, the paroxysms recurring at long intervals, is due to renal calculus usually, or the presence of a foreign substance, as blood, in the pelvis of the kidney. The pain is not only seated in the regions just indicated, but extends along the ureter, so that it extends from the loin to the front of the abdomen. It may persist for some time, at a point on either side of the umbilicus above or below it, or at a point on the surface of the abdomen opposite the brim of the pelvis. From thence the pain extends into the bladder either above the pubis (the hypogastric region), or into the testicle, or down the inside of the thigh. It may be in the loin and at the end of the penis at the same time, or lancinate along the whole urinary tract. In rare cases the pain is in the kidney of the healthy side. The pain of renal colic is always associated with frequency of micturition with or without pain during the passage of the urine. The character of the urine often points to the nature of the pain. The urine is usually bloody, and at first scanty; when the obstruction is removed it becomes copious. Renal pain or colic located in front of the abdomen must not be confounded with the pain of colic, either hepatic or intestinal. The pain is usually lower than in hepatic colic, extends along the course of the ureter, and is attended by symptoms referable to the urinary system and not the hepatic.

In tumors the pain may follow the course of the sciatic nerve, simulating sciatica. In pyelitis and hydronephrosis the pain is of a tearing character. The pain is variable in floating kidney.

Neuralgia. Neuralgia is a symptom of common occurrence in the course of Bright's disease. It may be due to anæmia, or be of uræmic origin. The occipital, supra-orbital, or trifacial nerve, or other nerves, may be affected. Anginoid seizures attended by pain are of frequent occurrence.

Frequency of Micturition. There are four causes of frequent micturition: (1) disease of the kidneys, the ureters, or the bladder, on account of irritability of the genito-urinary tract; (2) diseases in which

the amount of the urine is increased, demanding very frequent efforts to relieve the distention, as in diabetes; (3) diseases in which the urine is more concentrated, and hence causes more pronounced irritation, as in fevers, gout, or acute nephritis; (4) a reflex or pure neurosis.

Increased frequency of micturition on account of disease of the kidneys occurs in almost all organic affections of the genito-urinary system. It is seen in all forms of congestion and inflammation of the kidneys. In chronic nephritis it may not be noticed save that the patient is called upon to pass urine at night, arousing from sleep for this purpose. In some forms of nephritis the increased frequency may be due to increase in the amount of urine as well as increased sensitiveness of the organs. In the organic diseases it always occurs. The disease is not limited, however, to the kidneys. Disease of the ureter and disease of the bladder are also associated with this troublesome symptom. In its most aggravated and characteristic form it occurs in renal calculus, or when any foreign substance is located in the ureter or bladder. The frequency amounts to six, eight, or even a dozen times in an hour. It is often associated with tenesmus, the patient having a constant desire to urinate, at the same time passing but small amounts. This form of tenesmus is more frequent when the bladder or urethra is the seat of disease, and in renal calculus.

The Data Obtained by Observation. The Objective Symptoms.

The objective symptoms of diseases of the kidney are: (1) determined by physical examination of the organ; (2) derived by an examination of the urine; (3) due to impairment of the function of the kidney. Many of the latter symptoms are also subjective.

Physical Examination. Palpation and Percussion. The kidneys are situated in the right and left lumbar regions respectively, the left being a little higher than the right. They extend from the eleventh rib, or twelfth thoracic vertebra, to the third dorsal vertebra. The left kidney is in contact above with the spleen, and the right with the liver. The kidneys are enveloped in fat, more or less abundant; their distance from the anterior surface of the abdomen renders them inaccessible by palpation or percussion from that direction, and the thick dorsal and lumbar tissues, coupled with the relation of the kidneys with the organs, spleen and liver, which give a dull note on percussion, makes it difficult to outline the kidneys from behind. The best results are obtained by having the patient lie face downward, placing a cushion under the belly so as to make the lumbar regions a little more prominent. Strong percussion is required, and an artificial plessor and pleximeter are to be preferred. Percussion should be conducted with a view to marking the angle which the liver dulness and splenic dulness make with that of the kidney on the right and left sides respectively. The kidneys extend below the lower lines of liver and splenic dulness, and laterally for a width not greater than four inches. The difficulties in the way of outlining the kidneys by percussion are greatly increased in persons with much flesh, or when the abdominal walls are waterlogged, as they become in ascites, and practically it is impossible under such circumstances

to be sure of the boundaries of the kidneys. The colon must be empty to make the examination trustworthy.

Enlargements of the kidney are detected usually, first, by percussion, the width of the kidney being increased, and the percussion dulness extending, therefore, farther to the right or left, according as the right or left kidney is affected. As the causes which produce enlargements of the kidney which are sufficiently great to be detected by percussion do not, with rare exceptions, involve both kidneys at the same time, comparison of the two sides is of great value in the diagnosis.

Palpation of the kidney becomes possible when it is either enlarged or displaced. In the case of an enlarged kidney, the patient should lie upon his back or be turned slightly to the opposite side; one hand is placed beneath the kidney and upward pressure made while the other is pressed firmly and steadily from above or laterally toward the kidney. In this manner the kidney can be grasped between the two hands, its size estimated, and its physical characteristics as regards hardness, softness, fluctuation, and mobility determined. Enlargements are also detected by palpation of the abdomen. The renal tumor is usually two to three inches to either side of the median line, a little above the transverse umbilical line.

The diseases of the kidney attended by enlargement are *malignant tumors*, *tuberculosis*, *cysts*, *abscess*, *hydro-* and *pyonephrosis*, and *perinephritic abscess*.

In *abscess* of the kidney there is some fulness in the loin of the affected side. The kidney is felt to be enlarged, and is tender and painful. A tumor may be detected anteriorly. The diagnosis is made by a study of the cause (acute nephritis, pyæmia, impacted calculus in the ureter, erysipelas), and the detection of blood and pus in the urine, which is scanty; and by the constitutional symptoms. The progress of the case is usually acute. If the abscess is *tubercular*, tubercle bacilli can be detected in the purulent sediment of the urine, and there will be other foci of tuberculosis with a corresponding clinical history.

In *malignant tumors* of the kidney the surface is no longer smooth, and nodules may be felt.

In *pyonephrosis* the tumor is tense, smooth, and globular. Fluctuation may be detected. Tenderness is usually absent, and the course is slow and does not affect the general health so much as abscess. The pus may be discharged copiously from time to time, and the tumor be therefore diminished in size. The urine may be nearly clear at one time. Pyonephrosis arises secondarily to pyelitis, and often after the latter has lasted some time.

Hydronephrosis consists in a dilatation of the kidney pelvis with urine, which is prevented from escaping by obstruction of the ureter, either by the pressure of a tumor or by disease of the bladder or ureter itself. In time the kidney atrophies from the pressure and a large cyst forms. The tumor has the physical characters of pyonephrosis, but the history is different, and if there is any discharge it is free from pus. As in pyonephrosis, the tumor may become smaller, following a copious discharge—in this case of urine—or even may wholly disappear if the obstruction is removed. This sign is pathognomonic.

If obstruction continue to be absolute, the diagnosis must be made by the detection of a fluctuating renal tumor, the absence of fever and signs of suppuration, and by the result of exploratory puncture. The urine is usually free from changes.

It may be confounded with ascites if very large, but the hydronephrosis is rarely bilateral, and the fluid in it does not change its level upon change of position of the patient, as is the case with ascites. The history of the two conditions will be different.

An ovarian cyst can usually be traced into the pelvis, does not carry the colon in front of it, but is dull and even superficial on percussion, and leaves the loins resonant.

An *echinococcus* of the kidney presents the usual physical signs of such cysts. A fremitus may be detected or small cysts be found in the urine.

In the diagnosis of renal tumors in general it should be borne in mind that they are very rarely, almost never, movable with respiration. Unless too large they preserve their reniform shape, and press in front of them the ascending or descending colon, whereas ovarian tumors lie behind it. The position of the colon should therefore always be ascertained, and to this end it may be necessary to inflate it with air.

Perinephritis arises usually from extension of inflammation and suppuration from the kidney, but may be the result of strain, exposure to cold, or injury. Perinephritis may also be pyæmic, and occurs after infectious fevers. Gibney has reported twenty-eight cases occurring in children.

The swelling of a *perinephritic abscess* appears in the lumbar region of the side affected. It is rounded in form and doughy (Da Costa). Like other kidney tumors it is not affected by respiration. The usual signs of confined suppuration exist, and pulmonary or pleural complications may occur. As the abscess progresses the local signs of suppuration become more marked, the skin reddens, and pus may be discharged externally.

The most marked subjective symptom is pain, which may amount to agony, and is paroxysmal; soreness from restricted motion of the psoas muscle is apt to be complained of.

A tumor was present in the loins in sixty-five out of seventy-one cases analyzed by Fenwick, but generally did not manifest itself until the inflammation had made considerable progress. There is dulness on percussion even in the early stage, and later fluctuation. The general symptoms are vomiting, constipation, fever, and sometimes rigors. It is more common in males than in females (sixty-one males to thirty-nine females in Fenwick's cases); and is most apt to occur in persons who have suffered from renal calculi, pyelitis, or scrofulous kidney, often operations in the bladder and urethra, or when the patient has been subjected to injuries or strains of the loins, or to exposure to cold or wet when in a heated condition (Fenwick).

Floating kidney is best detected by palpation. It is recognized by its bean shape, its movability, the detection of the hilus and perhaps of the pulsation of vessels in it, and by the fact that it can be replaced. Palpation causes a sickening feeling, analogous to that experienced when

a testicle is compressed, but less in degree. A knee-chest position facilitates palpation. The value of relative percussion over the two kidney regions as a means of showing the absence of the kidney is much overrated. But the percussion will of course demonstrate that a body supposed from palpation to be the kidney is a solid organ.

The patient suffers from a feeling of lack of support in that region, which induces inaptitude and perhaps inability to work. The urine itself does not usually present any abnormalities.

Malignant tumors of the kidney, when primary, occur in a large number of cases in children. Twenty-five out of sixty-seven cases collected by Dr. William Roberts occurred in children under ten years of age. The most important symptoms are pain, hæmaturia, and tumor. The latter may grow with great rapidity and attain enormous size, filling the abdominal cavity and giving rise to pressure symptoms in surrounding organs. The growth occurs preferably anteriorly and downward toward the pubis, because there is less resistance in these directions. On palpation of the abdomen the tumor may appear smooth or irregular and undulated. As rapidly growing cancers are soft, the tumor frequently exhibits a certain degree of elasticity, which may be mistaken for fluctuation. It is immovable either by the hands or with respiration.

On percussion the resistance is increased and the note is dull, except in front, where the colon, which has been pushed forward, gives a tympanitic note. If the colon should be flattened out between the tumor and the abdominal wall it may be felt as a band stretching across the tumor, with dulness on percussion. In such a case inflation of the colon with air will be of great assistance in the diagnosis. Rare physical signs are pulsation and a blowing murmur.

Examination of the Urine.

1. Inspection. The urine in health is a clear yellow or amber-colored fluid, having a specific gravity of about 1020, and generally acid in reaction. It contains normally about forty-five parts in the thousand of solid matter, the principal part of which is urea—twenty-one and a half parts. The other solids are uric acid and its salts; certain extractives—creatin, creatinin, ammonia, hippuric acid, xanthin, hypoxanthin, sarcin, pigment, etc.; and chlorides, phosphates, sulphates, with their bases, soda, potash, lime, and magnesia.

The *volume* of urine passed in twenty-four hours is usually from forty to fifty ounces; but it may fall to thirty ounces or rise to seventy without the existence of disease. Women are believed to pass from five to ten ounces less than men. The volume is diminished when the skin is acting freely, as in warm weather, and when the bowels are loose; and, on the other hand, cold, nervous excitement, especially if it induce anxiety and fear, and constipation, all tend to increase the quantity secreted.

Color. The *color* of the urine is due largely but not wholly to urobilin, which is formed from the hæmatin of the blood. The color deepens when the urine is concentrated, as it becomes after a hearty meal

or exercise, especially in warm weather, and becomes paler when a large quantity is passed. The color frequently is changed in disease. In fevers the urine soon after being passed is apt to become *turbid* from the precipitation of urates, and the color varies from white, especially in children, to yellow, brown, or pink. When the precipitate settles, the supernatant urine may be high-colored and clear, or slightly opaque from some suspended matter.

The admixture of pus and chyle gives the urine a *milky* color. The urine may also be *yellowish-white* and turbid from phosphates, semen, sarcinæ, and bacteria.

The urine is *red*, reddish-brown, or "smoky" in acute nephritis, the color being due to blood. It is bloody in hæmaturia, cancer of the kidneys and bladder, and in injuries of the genito-urinary apparatus. A very red, clear urine is met with in concentrated urines containing a large amount of urates. The red color of the urine may be due to contained hæmoglobin, constituting *hæmoglobinuria*, or to urobilin, as in scurvy and pernicious anæmia. This occurs as the result of the action of certain poisons, such as chlorate of potash; in infectious diseases, such as scarlet fever, and in malarial fevers, also in a peculiar disease known as paroxysmal hæmoglobinuria.

Again, a *golden red* discoloration of the urine is common in jaundice; frequently the upper layers by reflected light have a greenish tinge.

Finally, a red color is produced by the internal administration of logwood and fuchsin.

A *yellow* color when opaque may be due to suspended phosphates and urates. Urine is also sometimes golden yellow or of a saffron color in jaundice, and from the effects of santonin, picric acid, and rhubarb taken internally. A yellow or yellowish-white turbidity may be due also to a mixture of pus and phosphates, and sometimes to semen, sarcinæ, and bacteria The urine usually becomes more or less opaque and yellow when it has undergone alkaline fermentation. Such a change occurs normally within a longer or shorter time after the urine has been passed. It is promoted by heat and exposure to air, and retarded by cold and exclusion from air. The urine whenever possible should be examined before this fermentation has occurred. Pathologically, in cases of cystitis, the urine is passed already in alkaline fermentation.

The urine is sometimes *chocolate-brown* when it contains blood and the blood has been acted upon by the urine, producing methæmoglobin.

Brown, *greenish-brown*, or *black* urine may result from contained bile salts; from indican; from carbolic acid, creosote, and tar used internally and externally; from the internal use of senna, and in cases where there are melanotic tumors. Senator injected melanin into human beings and obtained in four cases only a large indicanuria.

Urine is *pale* usually in proportion as it is copious in quantity. It is paler in those who are using milk or vegetable diet than in those who eat meats. Under the influence of nervous excitement, especially anxiety and the dread of an approaching ordeal, such as an examination, an abnormal quantity of very pale urine is secreted.

Pathologically, *pale* urine is characteristic of that passed in diabetes, chronic Bright's disease, and polyuria. Such urine is also secreted in

hysterical attacks, at the crisis of febrile diseases, and in anæmic conditions.

The Volume of the Urine in Disease. The volume may be increased, diminished, or unchanged in disease. It is *increased* principally in three diseases—diabetes mellitus, diabetes insipidus, and in the middle period of chronic Bright's disease, especially the interstitial form. In diabetes mellitus it sometimes exceeds thirty-two pints. It may be increased also in hypertrophy of the left ventricle, which induces greater pressure in the renal arteries as well as in the whole arterial system, and in cystic degeneration, and in double hydronephrosis.

The urine is *diminished* in acute nephritis and in the final stages of chronic nephritis; sometimes, also, it is diminished in the middle period of chronic nephritis, but usually it is here increased. All diseases which directly or indirectly impair the force of the circulation lessen the secretion of the urine. Hence the quantity is diminished in diseases of the heart muscle, and in valvular diseases not fully compensated; in emphysema and in chronic bronchitis. It is lessened also in cirrhosis of the liver. In febrile diseases the urine is scanty and high-colored, and sometimes it is almost suppressed (anuria).

The urine is sometimes *suppressed* in acute nephritis, such as follows scarlet fever, and in the final stages of all the organic affections of the kidneys—chronic nephritis, hydro- and pyonephrosis, etc. It may result (1) from the destruction of the secreting tissue of the kidney or interference with its nervous or vascular supply, or (2) from mechanical obstruction to the outflow of the urine. To the first class belong the cases of suppression occurring in acute and chronic nephritis, and the suppression from shock and collapse, whether occurring in the stage of collapse of yellow fever, cholera, and other grave febrile diseases, or from serious internal injuries.

Such suppression sometimes follows, also, slight operations on the urethra (urethral fever); or results from the internal administration of drugs the excretion of which occasions violent irritation of the kidney —cantharides, turpentine, and even the inhalation of ether. Clinically, suppression not due to obstruction is distinguished from the obstructive form by the character of the urine, which is usually not entirely suppressed, and by the more rapid course of the disease. The urine, according to Roberts, is either concentrated or it contains albumin, blood, and casts. Death or recovery results within a day or two. In the obstructive form, on the other hand, the urine which escapes past the obstacle is pale, watery, and devoid of albumin and casts.

Obstructive suppression is the result of the plugging of the ureter by a calculus when the opposite kidney is either absent or incapable of secreting. Or it results from the occlusion of the ureters by morbid growths, especially at the vesical orifices, from lateral pressure upon the ureters, or from some interference or malformation of the ureters or renal arteries.

Acute transient obstructive suppression occurs sometimes in persons with enlarged prostates who have drunk too freely of alcoholic beverages and perhaps have wound up a debauch by sexual intercourse.

The Density of the Urine. The average density of normal urine is about 1020. It may fall to 1015 or rise to 1025, depending upon the quantity of fluid and food taken, the condition of the atmosphere, especially as regards temperature, and upon the presence or absence of mental emotions. The specific gravity of the urine is tested by a urinometer graduated for degrees of density between 1000 and 1040. Only a reliable instrument should be used. As the density of the urine passed at different times of the day varies greatly, the urine for the whole twenty-four hours should be saved and a specimen of this tested.

The method of taking the specific gravity is very simple. A test-tube or graduate having a diameter of about one and a quarter inches and a length of six or seven inches is filled with urine to such a point that the lowest part of the urinometer floats clear of the bottom of the tube. The instrument must also float free of the sides of the tube. The specific gravity should then be read off from below, that is to say, by holding the tube up so that the level of the fluid is a little above that of the eye. Most urinometers are graduated for 60°, but in ordinary examinations it is not necessary to have the urine exactly at this temperature, but it should be allowed to cool after it has been passed, otherwise the specific gravity will appear to be too low.

In *disease* the specific gravity varies more widely than in health; it may fall to 1000 or 1005 in diabetes insipidus and chronic Bright's disease, and rise to 1060 or even higher in diabetes mellitus. As a rule, to which the urine in diabetes mellitus is the principal exception, the color is an index of the density, pale urine being of a low density and high-colored urine of a high density.

The density is increased when the urine is scanty in amount, whether as the result of fever, acute nephritis, large consumption of solid food, excercise, or free sweating. In all such cases the specific gravity rarely rises above 1035, and usually not above 1028 or 1030. When the specific gravity rises above 1035, and the urine is pale in color, the presence of sugar is to be suspected; and when it rises above 1040 sugar is almost certainly present.

The specific gravity is lowered by drinking copiously of fluids, by the effect of external cold, by a diet of vegetables and milk, and in general by the same causes that make the urine copious. Usually, but not always, a urine containing a large amount of albumin is of low density.

Pathologically, a low specific gravity is encountered in diabetes insipidus, in which it may fall nearly or quite to 1000; generally in the middle or quiescent period of chronic Bright's disease; in the crises of fevers; in obstructive suppression; in hysterical attacks, and in hydronephrosis.

Specific Gravity as an Index of the Amount of Solids. If the last two figures of the specific gravity be doubled the sum will represent the amount of solid matter in 1000 grains of urine. This is Trapp's method; the estimate is only rough, but it is useful. Of course, the urine for twenty-four hours must be used.

Reaction. The reaction of *healthy urine* is usually *acid*, but it may be neutral or slightly alkaline about two hours after a meal of mixed

food. The acidity is tested with litmus paper; the blue paper is turned purple or red by an acid, and the red paper is turned blue by an alkali. Violet paper is to be preferred, as it is suitable for showing both reactions, an alkali turning it blue and an acid red.

The acidity of the urine is *increased* in gout, lithiasis, acute rheumatism, diabetes, chronic Bright's disease, and as the result of the administration of vegetable or mineral acids.

The urine is *alkaline* as the result of alkaline fermentation in the bladder in cystitis; from the presence of much blood or pus; from prolonged immersion of the body in a cold bath; in debilitating diseases and in some cases of nervous dyspepsia, and as the result of the internal administration of alkalies.

Urinary Sediments. A white flocculent sediment composed of epithelium and mucus occurs normally in most urines after they have stood for some hours.

A dense sediment varying in color from that of brown sugar to pink or red, consists of amorphous urates. It dissolves upon the application of heat. A sediment usually resembling red pepper, but sometimes of a brown color, consists of uric acid. It can be proved to be uric acid by the murexid test. The suspected material is placed in a crucible or evaporating dish with a few drops of nitric acid. As heat is applied the uric acid or amorphous urate dissolves with effervescence. Heat is now kept up until the material is evaporated to dryness; it is then allowed to cool. If now it be touched with a glass rod dipped in strong ammonia a characteristic blue or violet color is produced. Uric acid is not usually so abundant as the sediment of amorphous urates; it sinks more rapidly, and is deposited from acid, high-colored urines.

A yellow or whitish sediment may consist of urate of soda.

A white sediment usually consists of phosphates, associated with which sometimes is a white sediment consisting of urate of ammonia, with or without pus. Such urines are alkaline. A white sediment may be due to uric acid, especially in children.

A yellowish-white sediment may consist of pus with or without mucus. If the urine be acid the sediment is loose and free to move, but when the urine is alkaline the sediment consists of a viscid, coherent mass, which can be drawn out into tough, stringy filaments.

A chocolate-brown sediment occurring in a reddish smoky urine consists of blood from the kidneys. Clots of blood come from the ureters, bladder, or urethra.

Odor. The odor of normal urine is sometimes spoken of as aromatic, but generally is sufficiently characteristic to be best described as urinous. When the urine is concentrated the odor is intensified, and may become unpleasantly strong, like that of the horse.

Certain articles of food, such as garlic and asparagus, cause the urine to smell of sulphides. Turpentine, both when taken internally and inhaled gives to it the odor of violets. The odor of copaiba and of cubebs can easily be detected in the urine of patients who are taking them.

In marked cystitis the natural urinous odor becomes more pungent, and is blended with a strong ammoniacal odor. When much pus is present and the urine has stood awhile the odor becomes putrid.

In diabetes mellitus the urine has a sweetish, hay-like odor. In diabetic coma the odor is sometimes that of chloroform, from the presence of acetone and diacetic acid. This odor, however, is more likely to be detected upon the breath.

2. **Chemical Examination of the Urine.** Examination of the urine by the unaided senses, which has been dwelt upon thus far, is simply preliminary to an examination by chemical methods and by instruments of precision, particularly the microscope.

Urea. Urea is freely soluble in water, and hence never appears as a sediment. It is the most important final product of nitrogenous disintegration in the body, and an index of the eliminative power of the kidneys. Usually the density of the urine increases in proportion to the amount of urea contained in it. The average daily amount of urea excreted by an adult man between the ages of 20 and 40 is about 500 grains. The urea, like the total volume of the urine, is subject to variations within the limits of health. It is increased after a meal, especially if it be rich in nitrogenous food; after copious infusion of liquids, and by a close atmosphere. On the other hand, fasting, free perspiration, a loose condition of the bowels, and a vegetable or milk diet diminish the quantity of urea. Again, the quantity varies with the age of the person. According to Ralfe, at five years the amount is 180 grains; at 12, 320; at 21, 535; and at 40 years, 555 grains.

A large man will excrete absolutely more than a small man, and a large muscular man will excrete relatively more than a fat man of the same height.

In disease, the urea is increased in fever and inflammatory diseases; in diabetes mellitus and insipidus; in malaria, pernicious anæmia, and after a crisis in pneumonia. It is increased also by certain beverages, as coffee, and by many drugs, especially those which act as hepatic stimulants.

It is diminished in all forms of nephritis, especially when uræmia results; in acute gout and chronic rheumatism; in diseases accompanied by emaciation and cachexia; and in leprosy, pemphigus, melancholia, imbecility, catelepsy, hysteria, and cholera (Saundby).

Estimation of Urea. For the methods employed in the exact quantitative estimation of urea, the student is referred to special works on the urine.

For ordinary clinical purposes the instrument devised by Professor Charles Doremus, and known as his ureometer, gives sufficiently accurate results. The principle upon which it is based is that urea when brought in contact with sodium hypobromite is decomposed and free nitrogen eliminated. The nitrogen evolved is the measure of the urea contained in the urine. The instruments are graduated so that each division of the scale represents one grain of urea per fluidounce of urine.

The hypobromite solution is made by dissolving 100 grammes of caustic soda in 250 c.c. of water and then adding 25 c.c. of bromine.

It is better, however, to have the hypobromite solution made fresh for each examination. This can readily be done by having a solution of caustic soda containing six ounces to a pint of water. It should be

kept tightly corked with a rubber or paraffined stopper. The caustic soda solution is poured into the long tube of the ureometer to the mark =, then one-tenth of its volume of bromine is introduced by means of a pipette, and sufficient water added to fill the long arm and the bend of the tube. The hypobromite solution should fill the tube completely, and any bubbles rising to the top of the tube be got rid of before the introduction of the urine. The pipette is then filled with the urine up to the 1 c.c. mark, any urine adhering to its surface being carefully wiped off. The pipette is introduced carefully so as not to compress the bulb until the point extends as high up as possible beyond the bend. The bulb is now compressed slowly, and bubbles of nitrogen rise to the surface of the long arm of the tube; when bubbles cease to be given off, the volume of nitrogen gas is read off as so many grains of urea per fluid-ounce of urine, or in milligrammes of urea in 1 c.c. of urine, according to whether it is graduated by the English or the metric system.

Detection and Estimation of the Chlorides. The presence or absence of chlorides is sometimes of diagnostic value. They are increased when absorption of exudations or transudations is going on, and in malarial fevers, diabetes insipidus, and Bright's disease. They are diminished or absent in pneumonia during its progressive stage, and in fevers. The chlorides can be detected and roughly estimated by an eight or ten per cent. solution of nitrate of silver. A few drops of nitric acid are first added to the urine to prevent the silver from also throwing down the phosphates. A single drop of the silver solution mentioned will precipitate the chlorides in a thick white lump, which falls to the bottom of the test-tube, provided they are present in the normal amount. If, on the other hand, they are diminished to one-tenth per cent. or less they will not be precipated in a lump or lumps, but as a white cloud which renders the whole solution opaque. If no precipitate whatever occurs the chlorides are absent.

Detection and Estimation of Serum-Albumin. Albumin is a very common, but it cannot be looked upon as a normal constituent of the urine, though its presence by no means necessarily indicates disease of the kidneys. The ordinary form is serum-albumin, but other proteids, as globulin, mucin, peptone, albumose, hæmoglobin, fibrin, and methæmoglobin are found at times. The most trustworthy tests for ordinary albumin (serum-albumin) are: boiling, with the addition of acetic acid or nitric acid; overlaying cold nitric acid with urine (Heller's test); and the picric acid test.

Boiling and Acetic Acid Test. A narrow long test tube is filled two-thirds full of urine and the upper third boiled thoroughly, and then a few drops of dilute acetic acid added. Any albumin present will be coagulated and appear as a white cloud contrasting strongly with the clear unboiled urine beneath it. When the albumin is moderate or small in amount it can be detected without difficulty by simply holding the test-tube up to the light. When there is a faint trace present it will be overlooked unless the tube be laid against a dark surface in such a way that the light falls upon it from above, in front and preferably slightly to one side. A cloud may escape detection when looked for by artificial light, but be distinct by daylight. Serum-globulin is

also thrown down by this test. But the globulin is not often present by itself, and its significance is not yet understood It may be detected in any urine, as Roberts points out, by diluting the urine with pure water, the urine then becoming more or less milky. It may be eliminated from urine by saturating the latter with sulphate of magnesia and filtering. The presence of serum-globulin in no way interferes with the test for serum-albumin.

If the urine is opaque from amorphous urates, it is unnecessary to filter them out; heat much below boiling will dissolve them, the precipitate from albumin occurring later.

If the urine is alkaline or faintly acid, phosphates will produce a cloud upon heating the urine; but they are instantly dissolved upon the addition of a few drops of acetic acid.

Mucin produces an opalescence upon heating with an organic acid, but Saundby declares that it coagulates not as albumin, but in the form of tiny filaments.

Boiling and Nitric Acid Test. This is preferred by many to the former. It is performed in a similar way; but the nitric acid so discolors many urines that the detection of small amounts of albumin is interfered with.

The Nitric Acid Test. This test, while not so delicate as the acetic acid test, is very simple and beautiful in its results. Cold nitric acid is poured into a test-tube to the depth of about an inch. The tube is then inclined to an angle of about 45 degrees, and urine allowed to flow gently down upon the acid by trickling along the side of the tube from a pipette or glass tube guarded by the finger. At the level of contact of the acid and urine a zone of white coagulated albumin forms. The thickness of the white zone is generally an index of the amount of albumin present. If there is barely a trace of albumin half an hour may be required to develop any opalescence.

A cloud of urates is sometimes thrown down and obscures the test. This cloud does not, however, begin at the level of contact and extend upward, but at the upper level of the urine and extends downward, and it is dissipated by heat.

Patients who are taking copaiba or cubebs pass a urine which gives a white ring at the point of contact with cold nitric acid, but heat diminishes the opacity, and the odor of the drugs named aids to their detection.

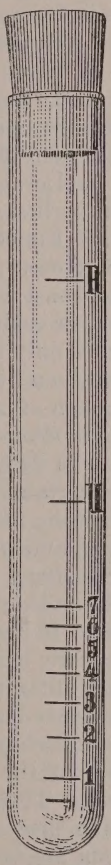
The Picric Acid Test. This is an extremely delicate test for albumin. A saturated solution of picric acid is allowed to flow down upon, and the upper layers slightly mix with, the urine which half fills a good-sized test-tube. At the level of contact an opaque white ring of coagulated albumin is formed. If no ring appears albumin is pretty certainly absent. Hence, the picric acid test is a valuable negative test. But, unfortunately, a ring is formed by peptone, mucin, and the presence in the urine of various alkaloids, particularly quinine. The latter disappears upon the application of heat, whereas an opalescence due to albumin becomes diffused throughout the whole urine.

Of the three tests, the first by boiling, with the subsequent addition of dilute acetic acid, is to be preferred. It is more delicate than the cold

nitric acid test, and it is free from the risk of burning one's fingers and clothes, which nitric acid always entails. The latter point is not an inconsiderable one for a physician who is obliged to make his urinary examination in his office whenever he can find time. The boiling and acetic acid test is to be preferred to the picric acid test as being just as easy of performance, and being at the same time free from the fallacies of the latter.

In all the tests for albumin mentioned a clear urine is necessary, especially when the amount of albumin is very small. This can be obtained by filtration when the opacity is due to pus, blood, mucus, and uric acid; and more effectively by the addition of liquor potassæ, heating, and filtering. If the filtrate in the latter case is not clear, a few drops of magnesian fluid (sulphate of magnesia, pure ammonium chloride and pure liquor ammoniæ, of each 2 drachms; distilled water, 2 ounces) as recommended by Hoffmann and Ultzmann, can be added, and the urine again warmed and filtered.

Fig. 92.



Esbach's albuminometer.

The *quantitative* estimation of albumin is of some importance. The most direct method is to coagulate the albumin by boiling, catch it upon a weighed filter, wash, dry, and weigh it. Such a process, however, consumes too much time for clinical purposes, and it is not faultless. An approximate estimation can be made by boiling the urine in a test-tube, allowing the albumin to settle, and then comparing the depth of albumin with the column of urine. In this way we can speak of urine containing one-tenth or one-quarter of its bulk of albumin.

Esbach has invented an albuminometer which gives better results. The solution used to precipitate the albumin consists of 10 grammes of picric acid and 20 grammes of citric acid, chemically pure, and dry, dissolved in 1000 grammes of hot water; any loss by cooling is to be made up by adding water sufficient to make one litre—1000 grammes. The urine is diluted with a definite amount of water if it contains too much albumin. The albuminometer is filled to the mark U, and from that level to R with the reagent. The tube is then corked with a rubber stopper, turned upside down ten times, so as to mix the urine intimately with the reagent, and then allowed to stand undisturbed for twenty-four hours. At the end of this time the level of coagulated albumin is taken according to the scale cut upon the glass. Each mark corresponds to one-tenth per cent. of albumin.

This estimation, as already stated, is not absolutely accurate. Nevertheless, if one always uses it, and in the same way, relative values will be obtained, and these are the most important in watching the progress of a case, as they give positive information regarding an increase or diminution of the amount of albumin in the urine. It scarcely need be said that the urine tested must be a sample of the whole twenty-four hours' urine.

ALBUMINURIA. Albuminuria is not indicative of disease of any one organ, nor does it point to any general pathological condition. It occurs as follows:

1. In diseases of the kidney: acute and chronic Bright's disease, amyloid disease, tubercle, cancer, abscess, and calculus.

2. In disturbances of the circulation: diseases of the heart and chronic pulmonary diseases, as emphysema; obstruction of the renal arteries or veins, cirrhosis of the liver, peritonitis, pregnancy, abdominal tumors; in passive congestions due to great weakness; in anæmia and Graves' disease.

3. In febrile and inflammatory diseases: in the eruptive and infectious fevers, and in rheumatism, diphtheria, pneumonia, and gout.

4. In blood diseases: purpura, leucocythæmia, and scurvy.

5. From the poisonous action of drugs: lead, turpentine, and others.

6. In nervous disorders: concussion of the brain and cerebral hemorrhage, epilepsy, tetanus, and delirium tremens; as Pye-Smith remarks, it is doubtful whether albuminuria is caused by the nervous disease.

7. Local extra-renal affections: pyelitis, cystitis, gonorrhœa, and leucorrhœa.

8. Functional. In young persons, particularly of the male sex, there occurs occasionally a small albuminuria after exercise, a special diet, or a cold bath. Albumin may be found after rising in the morning, or early after dinner or toward evening. On account of its occurrence only at certain times it has been called "cyclical" or "intermittent," and because there is no evident disease present, it is occasionally spoken of as "physiological" albuminuria.

Goodhart examined the urine of 1500 individuals and noted albumin in 272, or in 20 per cent. In 39 cases the albuminuria could not for certain be said to be due to disease of the kidney. Of these 39, 26 were males and 13 females. In 32 of the 39 cases it was temporary, and in most of them it had disappeared within forty-eight hours, or sooner. In 2 cases there were oxalates in the urine; in 1 hæmoglobinuria; in 8 leucorrhœal discharges and discharges from other parts of the genital passage (see division 7); and in 17 a markedly neurotic temperament. These last he thinks the most typical cases of intermittent albuminuria, while he regards the condition as less common than has been supposed.

A variety of functional albuminuria is due apparently to the irritation of the kidney produced by the excretion of oxalates and uric acid. The urine is of increased density, 1028, 1030 or higher, contains uric acid or oxalate of lime, or both, and cylindroids. Tube-casts are very uncommon. The albuminuria usually disappears under proper diet.

It is conceded that there may be albuminuria of renal origin without renal disease, but the diagnosis must be by exclusion, and can be reached safely only after extended observation. The most important elements in the diagnosis are the age of the patient, unimpaired general health, a specific gravity of the urine normal or above normal, the fact that the albuminuria is influenced by diet and exercise, and that it tends to disappear under suitable regimen. The prognosis is favorable.

PEPTONE. Peptone occurs in the urine in a variety of conditions, and hence not much diagnostic value can attach to its detection. According to Von Jaksch, its presence may indicate that a suppurative process exists; and when the diagnosis lies between epidemic cerebro-spinal meningitis and tubercular meningitis, the presence of peptonuria speaks for the former, but only when ulcerative processes in other organs, especially in the lungs, can with certainty be excluded. Exact tests for its detection are too elaborate for clinical purposes. The late Dr. N. A. Randolph suggested the following test, which is given by Tyson: To five c.c. of urine, which must be cold and faintly acid, add two drops of a saturated solution of potassium iodide and then three or four drops of Millon's reagent. If peptones or bile acids are present a yellow precipitate falls. If the yellow sediment does not respond to the test for bile acids it is due to peptone.

Picric acid when allowed to overlay urine containing peptone produces a white hazy ring which, unlike albumin, disappears upon the application of heat. If the patient has taken no vegetable alkaloids, particularly quinine, the ring described may be assumed to be due to peptone. Nitric acid and heat do not precipitate peptone.

MUCIN. Small quantities of mucin are present in all urines, being usually more abundant in women, from the admixture of the vaginal secretion. It is increased in catarrhal affections of the genito-urinary passages and of the bladder. It is thrown down by organic acids, but not by nitric acid.

According to Roberts, the best way to detect mucin is by means of a saturated solution of citric acid, employed in the same way as the contact method of applying the nitric acid test for albumin. A small quantity of the urine is first put in a test-tube and citric acid allowed to trickle along the sides of the tube until it forms a distinct layer below the column of urine. If mucin be present there will gradually appear an opalescent zone immediately above the layer of acid. Acetic acid mixed with one-third of its bulk of glycerin answers perfectly as a mucin test. Sometimes when mucin is very abundant the free addition of acetic acid without any precautions produces a marked milkiness of the urine. It is not re-dissolved by boiling.

BLOOD. Urine containing blood is usually red in color or reddish-brown and opaque, but it may be chocolate-brown if the blood is abundant and has been acted upon by the urine. It contains albumin.

Blood occurs in the urine from (1) *diseases of the kidney and urinary passages*, among which are Bright's disease, acute congestion of the kidney, renal calculus, cancer, tubercle; from ureteritis, cystitis, and urethritis, and from injuries; (2) from *general diseases*, such as the eruptive and intermittent fevers, scurvy, purpura, peliosis rheumatica, leucocythæmia, cholera; (3) from *adjacent organs*, as in menstruation and hemorrhage from the uterus; (4) from the *toxic action of drugs*—cantharides, turpentine, and other violent irritants of the kidney; (5) *vicariously*—occasionally menstruation fails to occur and hæmaturia replaces it. The same is true of bleeding from piles. Latour has reported a case of asthma which subsided suddenly upon the appearance of hæmaturia.

The chemical tests for blood are those for its coloring matter, and they will be referred to under Hæmoglobin.

Hæmoglobin. Hæmoglobin is of course present whenever blood is, but sometimes it occurs independently of hæmaturia. Thus it is found in grave infectious diseases, as the result of toxic action of drugs, such as carbolic acid, and in an independent disease known as paroxysmal hæmoglobinuria. A suitable test consists in adding one or two drops of fresh tincture of guaiac to about one drachm of urine; then shake the mixture and add a half-drachm of ozonic ether (*i. e.*, a solution of peroxide of hydrogen in sulphuric ether).

The same test answers for methæmoglobin and hæmatin.

Paroxysmal Hæmoglobinuria. The urine is bloody, or the coloring matter only is present. It is more frequent in males, and occurs in adults. It may be excited by a cold bath, or exposure to cold, and by exertion. It is sometimes associated with Raynaud's disease. The attacks come on suddenly, often preceded by chills. There is sometimes fever. Vomiting and diarrhœa occur with the hæmoglobinuria. Pain in the loins is sometimes complained of. The paroxysm may last a day or two, or two or three occur in the course of twenty-four hours.

Albumose. Albumose has been found in the urine in osteomalacia and diseases of the medulla of bone, in dermatitis, intestinal ulcer, measles, scarlatina, and mental diseases. Urine containing it does not respond at first to the heat and nitric acid test, but on cooling a precipitate forms which responds to the *biuret test.* (In this test the urine is first treated with caustic potash, and then a 10 per cent. solution of sulphate of copper added, drop by drop. If albumin be present the resulting peroxide of copper is dissolved, and the fluid becomes of a reddish-violet color.) The probability of the presence of albumose is strengthened if a turbidity occurs with the acetic acid and ferrocyanide of potassium test (acetic acid, specific gravity 1064, to which a few drops of a 10 per cent. solution of ferrocyanide of potassium has been added), and also with the biuret test, applied directly to the urine itself.

Detection and Estimation of Sugar. Next to albumin, sugar is the most important abnormal constituent of the urine. It is not present in normal urines in quantities that can be detected by ordinary clinical methods. The best tests for its detection are that by Fehling's solution and the fermentation test.

Fehling's Test. Fehling's solution is made as follows: Sulphate of copper, 90½ grains; neutral tartrate of potash, 364 grains; solution of caustic soda (sp. gr. 1.12), 4 fluidounces; water sufficient to make exactly 6 fluidounces. Two hundred grains of this solution, according to Roberts, are decomposed by one grain of sugar.

Certain precautions are necessary in the application of this test. 1. Any albumin present must be removed by boiling and filtration. 2. The Fehling solution is to be boiled first and the urine added to it; do *not* boil the urine first and then add the Fehling solution. Boiling the reagent first is a test of its stability; if a precipitate occurs the solution is unfit for use until soda or potash has been added to it and it has been filtered. As Tyson correctly says, a precipitate upon boiling the solution alone is more likely to occur when the Fehling solution has been

diluted with three or four times its bulk of water. 3. Prolonged boiling is to be avoided. Heat the solution to boiling and then add the urine; if no precipitate indicating sugar occurs until urine is added almost equal in bulk to that of the reagent, heat the mixture again to boiling and then set aside. 4. When the earthy phosphates are abundant it is well to get rid of them by adding liquor potassæ and filtering before applying the sugar test. 5. Changes in color occur from the presence of urea, uric acid, and extractives. These changes can be obviated when necessary by the method proposed by Seegen, who recommends repeated filtering through animal charcoal until the urine comes out colorless. The filter is then washed with distilled water and the sugar test applied to the water.

The method of applying the Fehling test is so clearly given by Roberts that one cannot do better than reproduce his words: "Pour some of the prepared test liquor, Fehling's solution, into a narrow test-tube to the depth of three-quarters of an inch; heat until it begins to boil, then add one or two drops of the suspected urine. If it be ordinary diabetic urine, the mixture after an interval of a few seconds will turn suddenly of an intense opaque yellow color, and in a short time an abundant yellow or red sediment falls to the bottom. If, however, the quantity of sugar present be small, the suspected urine is added more freely, but not beyond volumes equal to that of the test employed. In this latter case it is necessary to raise the mixture once more to the boiling-point. It is then allowed to cool slowly. If no sub-oxide has been thrown down when it has become cold, then the urine may with certainty be pronounced sugar-free." Again he says: "If no milkiness is produced as the mixture cools the urine may be confidently pronounced free from sugar, for no quantity above a fortieth of a grain per cent. can escape such a search, and any quantity below that is devoid of clinical significance."

The Fermentation Test. This is based upon the fact that yeast by fermentation separates sugar into alcohol and carbonic oxide. It is a certain but not very delicate test for sugar.

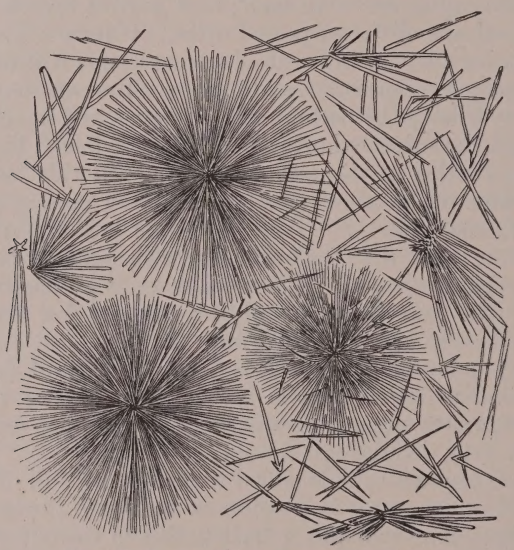
A small piece of yeast-cake is added to a test-tube full of urine. The tube is inverted under water in a saucer or beaker. If sugar is present in amounts larger than two and a half grains to the ounce, bubbles of carbonic oxide collect at the upper part of the tube after standing twelve hours in a temperature of about 90° F.

The Phenyl-hydrazin Test. Von Jaksch believes this test to be a very accurate one. About two grains of hydrochlorate of phenyl-hydrazin and three of acetate of soda are put into a test-tube half full of water. The contents of the tube are heated and the tube filled with the suspected urine. The tube is kept for fifteen or twenty minutes in boiling water, and then put in a vessel of cold water. When a large amount of sugar is present a deposit of yellow needle-like crystals is visible to the naked eye; but when only a small amount is present the sediment needs to be examined under the microscope. The crystals appear singly or in sheaves and fine radii. Yellow plates and brown balls do not indicate sugar. (Fig. 93.)

Quantitative estimation of sugar can be made with Fehling's solution by using a burette and measured quantities of urine and reagent. Tyson

recommends a method which answers very well for office use: One cubic centimetre of Fehling's solution is diluted in a large test-tube with four cubic centimetres of distilled water, and boiled. One-tenth of a cubic centimetre of the suspected urine is then added from a graduated pipette. Heat is then applied, the precipitate watched, and then another cubic centimetre added, and heat again reapplied until it is found, after proper subsidence, that all the color is removed from the cubic centimetre of Fehling's solution. If in doing this one cubic centimetre of urine has been added, it will have contained just one-half of 1 per cent. of sugar. If more than one cubic centimetre it will have contained less than one-half, but more than one-quarter per cent. If exactly two cubic centimetres are used, it will have contained exactly one-quarter per cent. If the quantity of sugar in the urine is large, the urine should first be diluted with a measured volume of water, this being regarded in the estimation.

FIG. 93.



Crystals of phenyl-glucosazon. (VON JAKSCH.)

When the quantity of sugar is relatively, large fermentation is the simplest and most trustworthy method. Roberts has shown that saccharine urine loses by fermentation one degree in density for every grain of sugar contained in an ounce of urine. For example, if the urine before fermentation had a specific gravity of 1040 and after fermentation a specific gravity of 1010, then the urine contained thirty grains of sugar to the ounce. In the application of this method about four ounces of the saccharine urine are put into a twelve-ounce bottle and a lump of German yeast about the size of a small walnut is then added to it. This bottle is closed with a perforated cork to allow the CO_2 to escape, and stood aside in a warm place to ferment. Beside it is placed a tightly corked four-ounce bottle filled with the same urine, but without any yeast. In about twenty-four hours the fermentation

will have ceased. The specific gravity of the fermented urine is then taken and also that of the unchanged urine. Every degree of loss of density represents one grain per ounce of urine.

Indican. An excess of indican is known as indicanuria. The substance is detected by several methods. Jaffe's test: Equal parts of hydrochloric acid and urine are mixed. By means of a glass pipette a solution of hypochlorite of soda is dropped into the fluid. An indigo-blue color is obtained. The hypochlorites must not be in excess. Weber's test is as follows: To 30 c.c. of urine and hydrochloric acid add 1 to 3 drops of dilute nitric acid. A quantitative examination is made by the colorimetric process of Salkowsky. A rough analysis is first made to determine the quantity of chlorinate of lime which causes the greatest abundance of indigo to unite with it. If the urine contains much indican, a small portion, as 2.5 to 5 c.c., is diluted with water to 10 c.c. If there is but little indican, 10 c.c. of the urine is used without dilution. Add to the fluid an equal quantity of hydrochloric fluid. To this add the amount of chlorinate of lime solution with which in the first test indigo formed in the greatest abundance. First neutralize the liquid with caustic acid, and then add enough carbonate of soda to make it alkaline. The indigo-blue is precipitated on filter. Repeatedly wash with water until the alkaline reaction disappears. The filtrate is dried and extracted by heating with chloroform until the latter does not take up its color. The chloroform extract is increased to a round number of c.c. by the addition of chloroform, and placed in a vessel with parallel sides. The intensity of its color is compared with a freshly prepared chloroform solution of indigo-blue of known strength. To one or other of these, chloroform is added until the tint of each is equal. The quantity of indigo-blue derived from the urine is determined and its percentage calculated from the constitution of the standard solution. Five to twenty milligrammes of indigo-blue are passed in twenty-four hours in health. When the mixture is boiled, a dark color is assumed. Allow the mixture to cool, and then shake up with ether. The indigo-blue is seen as a blue froth on the surface, while the ether is of a rose or violet tint. Indican is increased by animal diet—an increase which under other circumstances is pathological. Its presence is a sign of intestinal putrefaction. It may accompany a decomposition of albumin in cavities. It is present in empyema and in puerperal peritonitis. By detection of its presence in these, cavities due to pus may be distinguished from those due to other causes. Indican is increased in acute diarrhœa and in intestinal tuberculosis. Von Jaksch states that large quantities of indican in the urine imply that abundant albuminous putrefaction or putrid suppuration is in progress in the system. It must not be forgotten that in simple constipation indicanuria will often arise.

Bile Pigments and Bile Acids. Bile pigment or bilirubin occurs in the urine in cases of hepatogenic and hæmatogenic jaundice and in portal thrombosis.

Gmelin's test and its modifications are the ones usually employed. A small quantity of nitric acid, to which some nitrous acid has been added, is put into a test-tube and then gently overlaid with urine. If bile pigment is present a series of colors appear at the junction of the

two fluids—green, blue, violet and yellow. A green color (biliverdin) must be present to prove the existence of bile pigment.

The same test can be applied by placing a few drops of the acid upon one side of a plate and the urine on the other, and then allowing the two to run together. The play of colors takes place, as before, at the line of junction of the acids and urine.

Rosenbach's modification is an improvement. About 200 cubic centimetres of urine are allowed to flow through pure white filter paper, and then a drop of nitric acid is placed upon the paper saturated with the urine. The colors appear as before.

A very simple test consists in allowing a few drops of the acid to fall into a test-tube full of urine. If bile pigment is present a green color appears at the line of junction of the two fluids. If only small quantities of bile pigment are present, this test may fail to show it.

The tests for bile acids are either too elaborate or too unsatisfactory for clinical use.

Pus. Pus is found in the urine whenever there is suppuration or a catarrhal condition of the genito-urinary tract. Hence it occurs in abscess of the kidney, pyonephrosis, pyelitis, tubercle, cystitis, gonorrhœa, leucorrhœa, etc. It is relatively common in women, from a catarrhal condition of the vulva and vaginal mucous membrane, and hence in them is of less significance than it is in men. Urine containing much pus is slightly albuminous; but frequently pus cells are found in urine which gives no reaction for albumin.

The chemical test for pus is its conversion into a tenacious (gelatinous) glairy mass by boiling with caustic potash.

Acetonuria. An excess of acetone occurs in the following diseases: 1. In diabetes; 2, in cancer independent of starvation; 3, in starvation; 4, in certain psychoses; 5, in auto-intoxications; 6, in derangement of digestion; finally, 7, in fevers. In diabetes acetone indicates an advanced stage of the disease. Lieben's test is recognized by Von Jaksch: To several c.c. of urine a few drops of iodo-potassic iodide solution and caustic potash are added. If acetone is in excess, a large precipitate of iodoform crystals takes place.

Diaceturia. Diacetic acid is found in the urine in diabetes, in fevers, and in auto-intoxications. It is common with children in fever. It is of grave significance when in the urine of adults. Coma usually follows its passage in fevers and in diabetes. Test: Cautiously add a concentrated solution of perchloride of iron. Remove the filtrate if it is formed, and add more of the iron solution. Bordeaux-red color is developed. After the color appears divide the solution into two parts. Boil one part. If there is no change, test for acetone. The presence of this substance indicates that diaceturia is present.

Microscopic Examination of the Urine. Microscopic examination of the urine is chiefly concerned with the sediments, and these are conveniently divided into organized and unorganized.

The *organized deposits* in the urine are blood, pus, mucus, epithelium, casts, spermatozoa, micro-organisms, cancerous and tuberculous matter, entozoa.

The *unorganized deposits* are uric acid and its compounds, oxalate and carbonate of lime, phosphates, leucin and tyrosin, cystin and cholesterin.

Normal urine forms a slight sediment consisting of epithelium from different parts of the genito-urinary tract, principally from the bladder in males, and from the vagina and bladder in females. There are also some crystals of the different urinary salts, sometimes mucus and a few white blood-cells, and if the urine has stood awhile, especially if alkaline, more or fewer bacteria. It may accidentally contain extraneous matter derived from the vessel which contains it or from the air. (Fig. 94.)

FIG. 94.

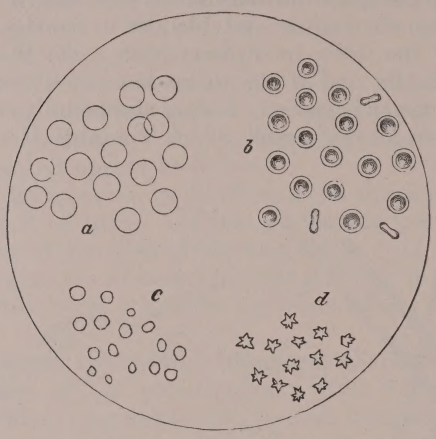
Extraneous matters found in urine: *a*, cotton fibres; *b*, flax fibres; *c*, hairs; *d*, air bubbles; *e*, oil globules; *f*, wheat starch; *g*, potato starch; *h*, rice-starch granules; *iii*, vegetable tissue; *k*, muscular tissue; *l*, feathers.

Organized Sediments.[1] BLOOD. If the blood comes from the kidney it is usually intimately mixed with the urine, which remains of a red or reddish-brown color, and contains possibly tube-casts and renal

[1] The use of the centrifugal machine in obtaining urinary sediment is of much practical value, and the writer highly recommends it. Not only is much time saved in completeing the examination of the urine, but all danger of its undergoing fermentation is avoided. The sediment can be

epithelium. The blood-cells appear singly, have frequently lost their hæmoglobin, and hence look like pale yellow disks. (Fig. 95.) Some-

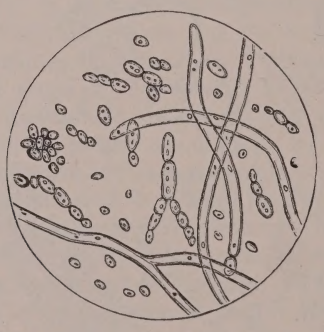
FIG. 95.



Blood corpuscles in urine. *a*, slightly distended by imbibition; *b*, showing their biconcave contour; *c*, shrivelled; *d*, serrated. (ROBERTS.)

times blood coagulates in the ureters, and long cylindrical plugs are passed, causing symptoms resembling those of renal colic. When blood comes from the bladder or neck of the bladder (fissure) there are symp-

FIG. 96.



Mould fungus. Sporules and thallus. (ROBERTS.)

toms of frequent micturition, of acute pain and tenesmus, and the blood is not intimately mixed with the urine. When from the neck of the bladder it often occurs in a few drops at the end of micturition,

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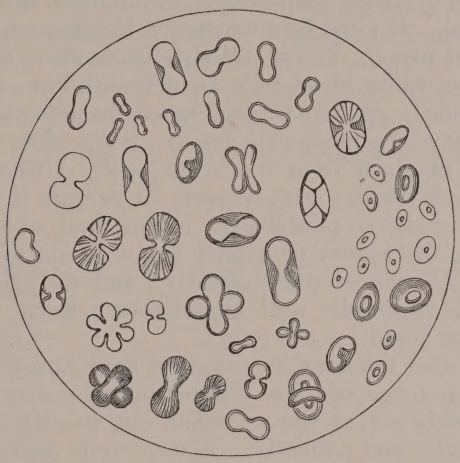
thrown down in three minutes' time by means of the machine without injuring casts or other contents. In using the machine, one of the glass tubes is three-fourths filled with the urine under examination and the handle revolved rapidly for about three minutes, or until three hundred revolutions have been made. The sediment will be found in a compact mass at the bottom, and is removed by a pipette and examined as detailed above.

For the preservation of urine until a sediment sufficient for examination is found, resorcin is the best antiseptic. A solution of fifteen grains to the drachm is made. One-half drachm will preserve four ounces of urine many days.

accompanied with great pain and a sense of faintness. Intermittent hæmaturia, according to Von Jaksch, points directly to calculus or tumor of the bladder.

Blood-cells when unaltered are unmistakable on account of their well-known biconcave appearance. When they have lost their coloring matter they appear as circular, very pale disks, with extremely faint outline and feeble refractive power. Absence of a nucleus serves to distinguish them from yeast spores (Fig. 96), and the latter, moreover, are often

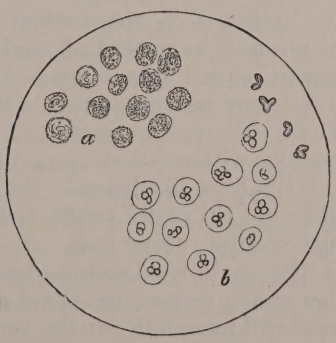
FIG. 97.



Dumb-bells and ovoids of oxalate of lime. (ROBERTS.)

oval in shape. They are less likely to be confounded with the ovoid and circular shapes of oxalate of lime crystals, because the latter are not common and can be seen usually in their more common forms as octahedra and dumb-bells in the same urine. (Fig. 97.)

FIG. 98.



Pus corpuscles. *a*, without reagents; *b*, after the addition of acetic acid. (ROBERTS.)

PUS. The sources of pus in the urine have been referred to already. The pus corpuscle is an opaque, spherical, granular cell, usually some-

what larger than a red blood-cell. In dilute urine or upon the addition of water it swells sometimes to twice its original size. At the same time it becomes less granular, and two, three, or four nuclei may appear. In concentrated urines the pus cell is small. The addition of acetic acid also, causes it to swell and brings out the nuclei more distinctly and rapidly. Sometimes the pus cells are discrete, sometimes in dense clumps, and sometimes nothing but a dense mass of pus cells appears in the field of the microscope. (Fig. 98.)

It cannot be decided from microscopic examination whether a cell is a pus corpuscle, a mucus corpuscle, a white blood-cell, or an inflammatory leucocyte. It must be a matter of inference from the general characters of the urine. If red blood-cells are also present, the probability of finding white blood-cells is increased, but pus cells are not necessarily excluded. So, too, if much mucus be present in the urine, the doubtful cell may be a mucus corpuscle. Some clue to the source of the pus can be obtained from the urine itself. Urine containing pus from the kidney is usually acid, whereas in cystitis it is alkaline and almost always contains phosphates, mucus, and abundant bacteria. Again, pus from the kidney or kidney pelvis is apt to vary greatly in amounts, or be discharged intermittently; and the urine when filtered free of pus cells is usually still albuminous. Renal epithelium and casts also may be found.

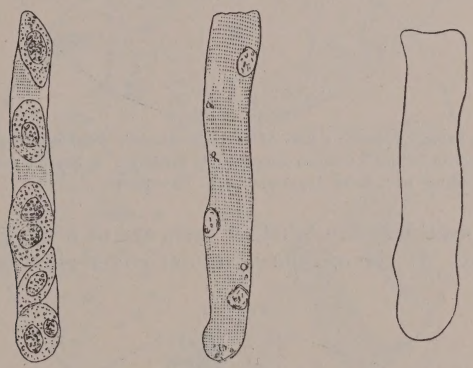
Casts. Casts are the most important of the urinary deposits. They vary greatly in number and size. Sometimes in acute nephritis they form a considerable part of the sediment, but usually they have to be sought for carefully and patiently. A few words as to the method of examining for them may not be superfluous.

Six or eight ounces of the urine to be examined should be allowed to settle in a bottle as soon after being passed as possible. The bottle should be tightly corked, because urine exposed to the air decomposes very quickly; and it should be sent to the person who is to examine it as soon after being passed as possible in order that an examination can be made before fermentative changes spoil it for trustworthy analysis. After standing twelve, or preferably twenty-four hours, nearly all the solid matter will have collected in the bottom of the bottle. The supernatant clear fluid can now be poured off and the lower portion of the urine and the sediment poured into a conical subsiding-glass. If the urine is febrile there may be by this time a large deposit of amorphous urates which will obscure the search for casts; they may be dissolved by gentle heating without destroying the casts, and the clear urine again allowed to settle for a few hours. So, too, if phosphates are abundant, they should be got rid of by gentle heating and acidulation with two or three drops of dilute acetic acid.

After the urine in the conical subsiding-glass, which will not now amount to more than an ounce or two, has stood for a few hours, any casts that may be present will have fallen to the bottom. If the urine is very concentrated (1030 or more), epithelium, blood, and casts will be suspended longer; hence it may be well to dilute the urine before allowing it to settle.

A glass tube with an internal diameter of about one-eighth of an inch, and with one end drawn out fine, is the most convenient thing for collecting the sediment. The ordinary glass pipette with a rubber suction-bulb at one end, commonly known as a "medicine dropper," sometimes answers admirably. If the common glass tube is used, the forefinger of the right hand should be placed over the open upper end, and the fine lower end passed down to the bottom of the glass. The finger is then removed sufficiently to permit a few drops to be sucked in. The same result is attained if the finger is entirely removed as soon as the point of the tube reaches the bottom of the conical glass; but in that case more than the lowest layers of the sediment or urine are sucked up, and hence all but a few drops should be allowed to flow out when the tube is removed from the urine. In this way the drops preserved for microscopical examination will contain the sediment from the very bottom of the glass; and in this sediment, in pale urines free

FIG. 99.



Epithelial and hyaline casts.

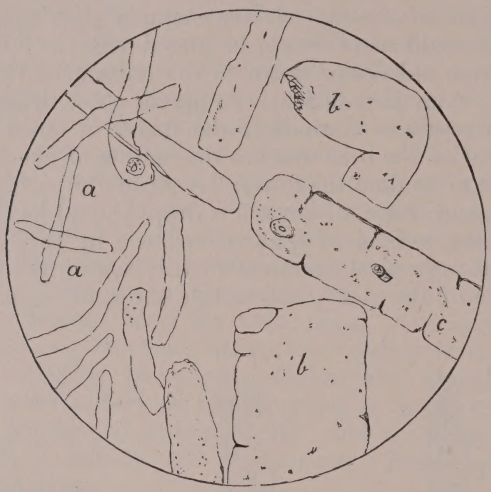
from much urates, phosphates, and pus, the casts will be found, if any are present in the urine. It is most important to examine the bottom layers of the sediment when the latter is scanty or when phosphates or urates have begun to precipitate after the urine has been standing some time. If the urine is already cloudy with phosphates, urates, or pus when it is put aside to settle, any casts that may be present will be carried down with the heavier sediment and will be found intimately mixed with it, or even on top of the other sediment.

The few drops preserved for microscopic examination are now deposited on several slides, covered with a cover-glass in the usual way, and examined carefully for casts under a power of 200 or 300 diameters. Casts may be numerous, so that nearly every field contains one or more, or they may be very few, not more than one or two being found on a slide.

When great importance attaches to the examination it is better to use a square cover-glass, so that starting, say, from the upper left-hand corner, and moving the slide slowly, keeping the upper edge in view, until the upper right-hand corner is reached, and then taking a field

lower down and running the slide in the opposite direction until the left edge is again reached, and so on, the whole slide can be examined,

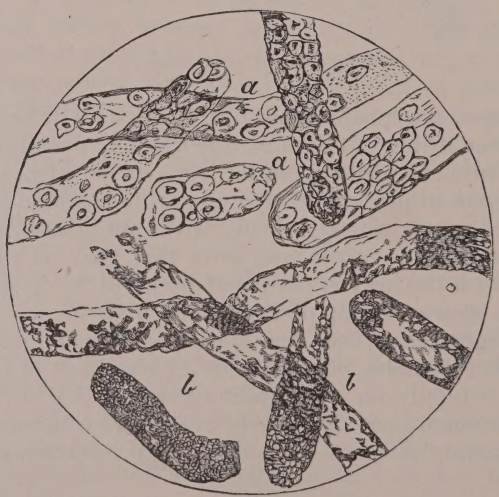
FIG. 100.



Hyaline and waxy casts. *a*, From a case of chronic Bright's disease of eight months' duration. *b*, From a case of chronic Bright's disease (large white kidney). *c*, From a case of chronic Bright's disease (contracted kidney with fatty degeneration). (ROBERTS.)

and one can positively say whether casts are or are not present in that particular slide. If the ordinary circular cover-glass is used, the same

FIG. 101.



a, Epithelial casts. *b*, Opaque granular casts, from a case of acute Bright's disease. (ROBERTS.)

field may be re-examined several times and other parts of the slide never seen. All the pipettes used in examining urine must be kept clean.

They should be allowed to stand in water which is frequently changed, and carefully rinsed in running water before being used.

Tube-casts usually indicate acute or chronic nephritis; but they are *sometimes* found in cases of renal calculi; in icterus, usually without albuminuria; in diabetes, and sometimes in secondary congestion of the kidney.

Several varieties of casts are found. 1. *Hyaline casts*, as their name implies, are clear, translucent bodies, which refract light so slightly that they are easily overlooked. They have well-defined margins, the ends being frequently rounded; they are rarely very long, and are straight, or but slightly bent. They are rarely equally translucent throughout; at some part more or less granulation will generally be found. In size they vary in diameter from that of a white blood-cell to six or eight

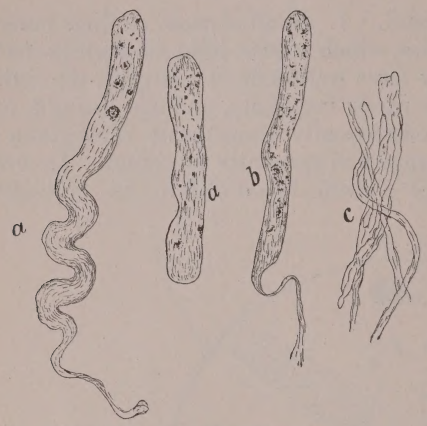
FIG. 102.

a, Fatty casts; *b* and *c*, blood-casts; *d*, free fatty molecules. (ROBERTS.)

times as large. They can be stained and so rendered more distinct by allowing a drop of gentian-violet solution to flow in under the edge of the cover-glass. (Figs. 99, 100, *a a*.) 2. *Granular casts* are hyaline casts which appear granular either from some deposit on their surface or from a granular change of the cast itself. When the granulation does not interfere with the translucency, the casts are described as "pale" or "slightly" granular; and when they become very dark, so as to resemble closely a blood-cast, they are called "dark" or "opaque" granular casts. (Fig. 101, *b*.) 3. *Waxy casts* appear to the eye to be more solid in structure than the hyaline casts; they also appear more cylindrical in form, are more or less yellow in color, and are apt to be larger than hyaline casts. (Fig. 100, *b*, *c*.) 4. *Fatty casts* are hyaline or faintly granular casts on which are deposited in spots minute oil-drops. These are sometimes called "oil-casts" if the oil-drops are very abundant. (Fig. 102, *a*.) 5. *Blood-casts* are either made up of a mass of blood-cells pressed together into a cylindrical shape, or, more

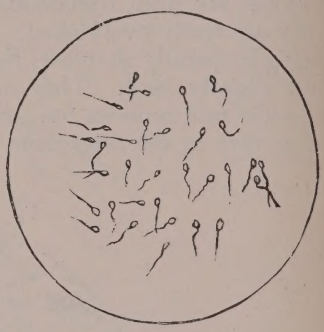
frequently, a hyaline cast is studded with blood-cells. (Fig. 102, *b*, *c*.) 6. *Epithelial casts* seem sometimes to be composed entirely of epithelial cells closely packed together. Such casts are relatively rare, and very beautiful. Ordinarily, just as in the case of blood-casts, an epithelial

FIG. 103.



Cylindroids. (DR. ALFRED STENGEL.)

FIG. 104.



Spermatozoa. (ROBERTS.)

cast consists of a hyaline cast more or less covered with renal epithelium. (Fig. 101, *a*.) 7. Dr. George Johnson has described casts composed of *pus* corpuscles. In two cases in which they were found in the urine the patients were found at autopsy to have multiple abscesses of the

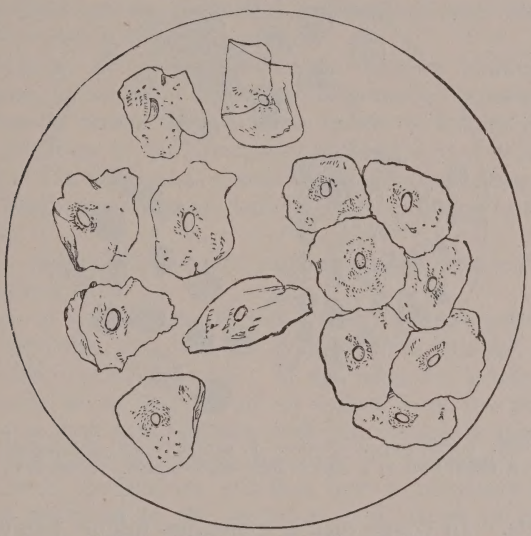
FIG. 105.

Human semen. *a*, Spermatozoa; *b*, cylindrical epithelium; *c*, bodies enclosing lecithin granules; *d*, squamous epithelium from the urethra; *d′*, testicle cells; *e*, amyloid corpuscles; *f*, spermatic crystals; *g*, hyaline globules. (VON JAKSCH.)

kidney. 8. *Cylindroids* are very common. In general appearances they resemble hyaline casts; but they are apt to be much longer, to be bent, twisted, or split, and to have, on close examination, a striated or finely ribbed appearance. Moreover, the diameter of the cast frequently varies greatly at different points, sometimes it appears constricted in several places, and in other cases one end tapers off into a thread. Often they consist of fine, narrow ribbon-like threads. (Fig. 103.)

SPERMATOZOA. Spermatozoa are easily recognized by their tadpole shape and by the vibratile motion of their long delicate tails. They are found in the urine of both sexes after sexual intercourse. (Fig. 104.)

FIG. 106.



Vaginal epithelium in the urine. (ROBERTS.)

Many continent men have occasionally nocturnal emissions, accompanied by erections and erotic sensations. These cannot be looked upon as abnormal, and they are compatible with robust health. There

FIG. 107.

a a', Pavement epithelium from urinary sediment; *b b' b''*, bladder epithelium; *c c' c'' c'''*, renal epithelium; *d d'*, fatty degenerated renal epithelium; *e*, *h*, bladder epithelium. (VON JAKSCH.)

are other persons, neurotic, anæmic, and generally constipated in habit, who have emissions at night two or three times a week, of which they are unconscious until after they wake and find themselves wet. Semen may also be lost during micturition and defæcation, especially when much straining is required. Such a condition (spermatorrhœa) is abnormal. It is due to general nervous and muscular relaxation, associated with

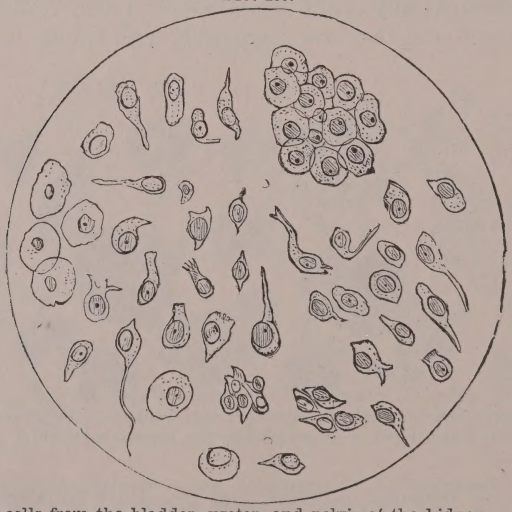
nervous dyspepsia and anæmia, and aggravated by sedentary life, constipation, and the reading of salacious literature or the cultivation of

FIG. 108

Renal epithelium. *a*, Natural appearance. *b*, Atrophied and disintegrated renal cells. *c*, Renal cells in a state of fatty degeneration. (ROBERTS.)

erotic thoughts. In young men it sometimes follows habits of masturbation, which have been broken up but have left behind a hyperæsthetic

FIG. 109.



Epithelial cells from the bladder, ureter, and pelvis of the kidney. (ROBERTS.)

condition of the prostatic portion of the urethra, with or without dilatation of the orifices of the ejaculatory ducts; or a stricture of gonorrhœal origin may be its cause. Students and overworked and

overstrained business and professional men are the ones most frequently affected.

However caused, the condition is apt to beget a most distressing state of despondency, in which the patient imagines all possible ills and is liable to drift into an hysterical, melancholic, even suicidal frame of mind, and so falls a victim to quacks.

Epithelium. Epithelium from the kidney, bladder, and genito-urinary passages occurs in the urine. Epithelial deposits in male urine are very scanty, unless there is some disease of kidney or bladder, or a catarrhal condition of the prostratic urethra, such as is left from an old gonorrhœa. On the contrary, considerable epithelium may be normally present in the urine of women, being derived principally from the vagina and bladder.

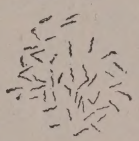
Vaginal epithelium consists of large flat pavement cells, and is readily distinguished.

The type of epithelium of the kidney, kidney pelvis, ureter, and bladder is the same, and it is not possible to distinguish with certainty the cells which come from each. If the cells are scanty, Von Jaksch thinks this fact to be in favor of an origin from the ureter. He has found them in moderate quantity and superimposed upon one another.

Renal cells resemble closely the oval or polygonal cells from the deeper layers of the bladder, but they have a relatively larger nucleus.

Fat. In addition to the presence of oil-drops in association with fatty degeneration of the kidney and its epithelium, oil is found occasionally in the urine of those who are taking cod-liver oil, and in calculous disease of the pancreas. Tyson suggests that it may come from cystic cheesy degeneration of the kidney.

Fig. 110.



Vibriones in urine. (Roberts.)

Lipuria. In chronic nephritis, in phosphorus poisoning, and in diabetes mellitus fat is found, as well as in chyluria. The urine is turbid, but clears when agitated with ether. The fat may be separated by a sedimentator, and can be recognized by its refracting properties.

Chyluria. This is a more or less milky condition of the urine, due to the presence of fat, which probably gains entrance to some part of the urinary tract by rupture of the lymphatic vessels. A case has been reported by Saundby in which a young unmarried girl, having been pregnant, compressed her abdomen so much in order to conceal her condition that œdema of the legs, thighs, vulva, and lower part of the abdomen resulted. After her confinement the urine became milky, and remained so for many days. It contained fatty matters, cholesterin, but no albumin or sugar.

Fat and albumin appear at the same time in some diseases. They

recur at long intervals. Red and white corpuscles are also found in small amounts. The urine coagulates on standing, or gelatinizes. It is due to the invasion of the urinary tract by the filaria sanguinis hominis, the embryo of which is found in the urine.

Parasitic chyluria is due to the filaria sanguinis hominis, whose embryos obstruct the lymphatics.

Entozoa. The most common is the *echinococcus* or *hydatid.* When this infects the kidney or urinary vessels hooklets and even cysts have been passed in the urine. The disease is, of course, extremely rare in this country.

The *filaria sanguinis hominis*, which causes parasitic chyluria, is occasionally found in the urine. (See Filaria.)

The *Bilharzia hæmatobia* sometimes lodges in the urinary tract and causes hæmaturia. It is peculiar to Egypt.

Intestinal worms may creep into the bladder through fistulous or other openings, and be discharged through the urethra.

Fig. 111.

Various forms of uric acid crystals. (Finlayson.)

Micro-organisms. Normal urine contains no micro-organisms at the time it is voided. As the result of exposure to the air, however, they may develop in great abundance. The non-pathogenic organisms found are classed as mould fungi (hyphomycetes), yeast fungi (blastomycetes), and fission fungi (schizomycetes).

Mould fungi, according to Von Jaksch, are rarely found in foul normal urine. Yeast fungi are also rare in normal urine. Fission fungi are found in urine undergoing ammoniacal decomposition.

Sarcinæ, usually smaller than those of the stomach, are occasionally met with—especially, according to Roberts, when there is some disorder of the urinary organs, renal pains, painful micturition, cystitis, etc.

Under the name *bacteriuria* Roberts and others have described cases in which the urine at the time of being voided contained bacteria. He makes four groups : (1) Cases in which the presence of bacteria is asso-

ciated with incipient putrefactive changes in the urine; (2) cases associated with ammoniacal fermentation of the urine; (3) cases in which the common forms of bacteria are present without decomposition of the urine; and (4) cases in which micrococcus chains are voided in the urine.

FIG. 112.

Amorphous urate deposit. (ROBERTS.)

The pathogenic organisms which are more or less closely associated with infectious diseases, septic processes, and tuberculosis, are found at times in the urine, and can be demonstrated by the proper staining methods.

FIG. 113.

Urate of soda.
a a. From a gouty concretion; *b b*. Artificially prepared by adding liq. sodæ to the amorphous urate deposit. (ROBERTS.)

FIG. 114.

Hedgehog crystals of urate of soda, spontaneously deposited from the urine of a child. (ROBERTS.)

MORBID GROWTHS. The urine very rarely contains the elements of morbid growths. Von Jaksch says he never has found them in any way reliable in the case of tumors of the kidney. The detection of cancer cells or pigmented cells, such as occur in melanotic cancers, may confirm the diagnosis, if the clinical symptoms point to cancer. Tumor elements are most likely to be found in ulcerating tumor of the bladder.

Unorganized Sediments. *Uric Acid.* Uric acid is present in small quantities (eight to ten grains a day) in normal urine. It is *increased* in febrile and wasting diseases, such as phthisis; in diseases of the liver and spleen (leukæmia), and in malarial fever, diabetes, scurvy, rhachitis, and following an attack of gout. Excessive use of milk is said sometimes to increase it. Its excretion is also increased by certain drugs—colchicum, corrosive sublimate, and euonymin.

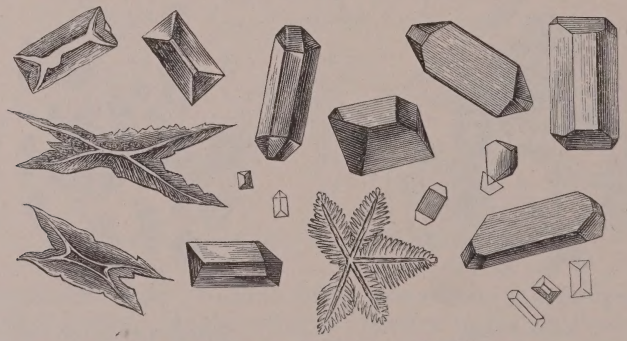
FIG. 115.

Urate of ammonia spontaneously deposited.

a. Spheres and globular masses; *b*. Dumb-bells, crosses, rosettes. (ROBERTS.)

It is *diminished* in anæmia, chlorosis, during a paroxysm of gout; in chronic nephritis; by certain drugs—large doses of quinine (Ranke), caffein, sodium chloride and sodium carbonate, lithia, and iodide of potash.

FIG. 116.



Various forms of triple phosphates. (FINLAYSON.)

According to Roberts, a deposit of uric acid occurring some twelve to twenty hours after the urine has been passed has no pathological significance. If the deposit occurs within three or four hours after it has been passed it is certainly not natural: it is frequently observed in convalescence from febrile complaints, especially articular rheumatism; also in the middle periods of chronic Bright's disease, in chorea, in certain types of diabetes, and in enlargement of the spleen. If, however, the uric acid is precipitated before the urine cools, or immediately afterward, there is a liability that the same precipitation may occur within some part of the urinary passages, and so form a calculus.

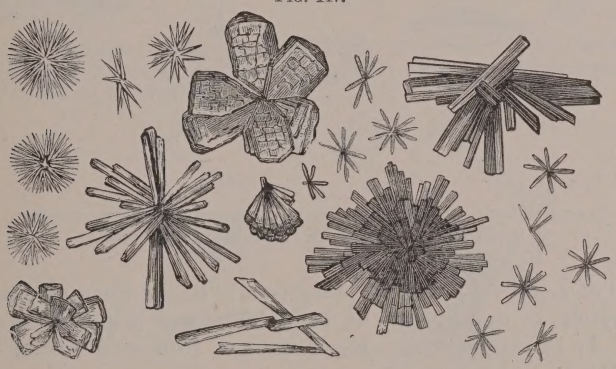
Urates. Amorphous urates appear under the microscope as opaque granular particles, which dissolve upon heating, and respond to the murexid test. The deposit is more or less dense, and is sometimes arranged so as to resemble granular casts. (Fig. 112.)

Urate of Soda appears as spherules or globules from which project short spines, either straight or curved. It occurs most frequently in concentrated acid urines, such as are passed by children with acute febrile diseases. (Figs. 113 and 114.)

Urate of Ammonia resembles the urate of soda, except that it has no spines. It is associated frequently with phosphatic deposits, and is precipitated from alkaline urines. Sometimes it appears in the shape of dumb-bells. (Fig. 115.)

Phosphates. Phosphates appear in the urine as ammonio-magnesium phosphate, and as the crystalline and amorphous phosphate of lime.

FIG. 117.



Crystalline phosphates. (FINLAYSON)

They are precipitated in alkaline or faintly acid urines, which produce a cloud upon being heated; the cloud is distinguished from albumin, as already pointed out, by disappearing when the urine is acidulated with acetic or nitric acid. Ammonio-magnesium phosphate is easily recognized by its rhombic prisms—"coffin-lid" shape. Other shapes are produced by modification of the primary one, chiefly by bevelling of the edges and hollowing out of the sides. These crystals are usually large, and are frequently found together with amorphous phosphates, bladder epithelium, and pus, in cases of cystitis.

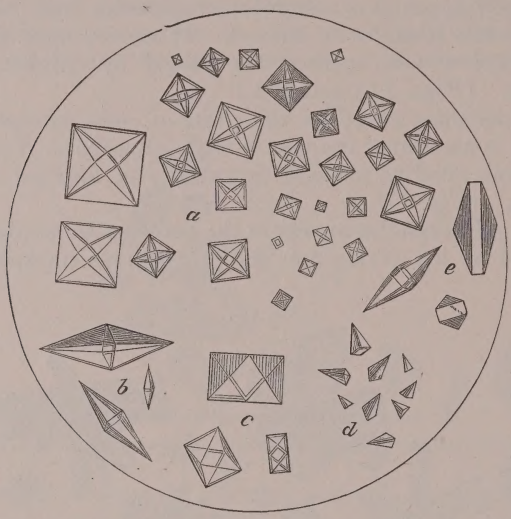
Amorphous phosphate of lime consists of fine granular particles, much resembling amorphous urates, but distinguished from them by not disappearing upon the application of heat, but instantly dissolving when the urine is acidulated.

Crystalline phosphate of lime is a rare deposit. It is found as rods or needles, and occasionally grouped together in the form of stars, sheaves, or bundles.

According to Roberts, this deposit in quantity is an accompaniment of some grave disorder. He has found the stellar phosphates in cancer of the pylorus, once in phthisis, and more than once in patients

exhausted by obstinate chronic rheumatism. It may, however, occur in health, when the urine is rich in lime and its acidity greatly reduced.

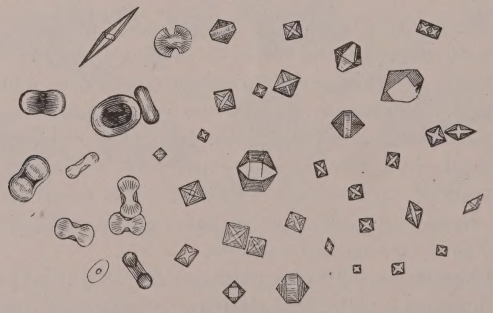
FIG. 118.



Oxalate of lime. *a*, *b*, *c*, Octahedra in various positions; *d*, pyramids; *e*, pyramids with intervening square bases. (ROBERTS.)

Oxalate of Lime. Oxalate of lime occurs in the form of small octahedral crystals, or more rarely as dumb-bells, and in the form of ovals or disks. It is precipitated, almost always, from acid urines. (Figs. 118 and 119.)

FIG. 119.



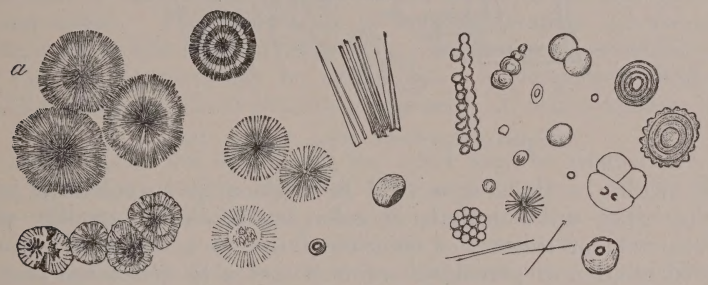
Less common forms of oxalate of lime crystals. (FINLAYSON.)

According to Beneke, oxaluria has its proximate cause in an impeded metamorphosis, an insufficient activity of that stage which changes oxalic acid into carbonic acid.

When oxalates are constantly found in the urine a condition of profound hypochondriasis is found to exist, but it has no necessary relation to the oxaluria.

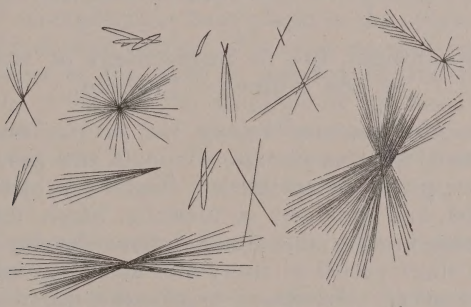
Oxaluria. An increase of oxalate in the urine is found in diabetes, especially when there is diminution in the amount of sugar. It is in excess in certain forms of indigestion. Its constant passage may be attended by pains in the back and loins. Flatulent and nervous dyspepsia usually accompany the increase, and, with neurasthenia, are common.

Fig. 120.



Crystals of leucin (different forms). (Crystals of creatinin chloride of zinc resemble the leucin crystals depicted at *a*.) The crystals figured toward the right consist of comparatively impure leucin. (From Charles: *Chemistry*.)

Fig. 121.



Tyrosin crystals. (From Charles: *Ibid.*)

Cystin. Cystin occurs in the form of hexagonal prisms, either as irregular masses, or superimposed one upon another, so as to form truncated pyramids. It is a very rare sediment, but appears to be most common in children and young male adults. Several members of the same family have been known to pass it. Its chief clinical significance arises from the fact that it is rarely the basis of calculi.

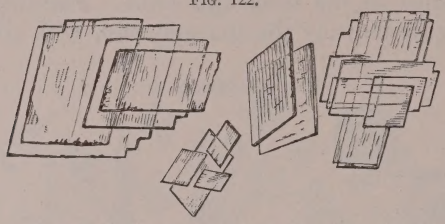
Leucin and Tyrosin. Leucin and tyrosin are generally described together, though the former is not spontaneously deposited from urine. It appears in the form of spheres which refract light strongly and have a radiating arrangement. (Fig. 120.)

Tyrosin has been found as a sediment, of a light greenish-yellow color, in typhoid fever and acute yellow atrophy of the liver. It appears in the form of tolerably long needle-like crystals, or as bundles and sheaves. Frerichs attaches great importance to leucin and tyrosin in the diagnosis of acute yellow atrophy of the liver. (Fig. 121.)

Cholesterin. This occurs at times in fatty degeneration of the

kidneys, jaundice, chyluria, diabetes, and, according to Pöhl, in the urine of epileptics treated with bromide of potash. (Fig. 122.)

FIG. 122.



Crystals of cholesterin. (From CHARLES: *Ibid.*)

MELANURIA. Melanin is held in solution or suspended in small granules. The urine is dark in color, but blackens intensely when sulphuric acid or tincture of chloride of iron is added to it. A concentrated solution of perchloride of iron serves to detect the presence of the substance. A few drops added to the urine turn it gray. If a few drops more are added, the phosphates are precipitated along with the coloring matter. Both are dissolved by an excess of the iron solution. Melanin is usually found in cases of melanotic carcinoma.

Objective Symptoms due to Impairment of the Function of the Kidney. Uræmia.

To this class of symptoms belong the various manifestations of uræmia. Diseased kidneys do not eliminate the products of tissue waste which are poisonous materials. The toxic matter is retained within the blood, and produces a toxæmia, which may be acute or chronic. In *acute uræmia* the manifestations develop suddenly, and continue but a short period of time, with alarming active symptoms until death or recovery. In *chronic uræmia* the onset is gradual. The manifestations may be limited to one or two conditions, as headache or morning nausea, or they may include the more pronounced forms of uræmia.

CEREBRAL SYMPTOMS. 1. *Headache.* The pain is situated in the occipital region, and may extend down the neck. It is severe and of a bursting character. It may be associated with giddiness. In both acute and chronic nephritis it is often the first manifestation. It may be associated with eye symptoms. It may be present on waking, and continue only through the morning hours. In acute uræmia it persists throughout the attack. Numbness and tingling of the fingers are often complained of at the same time.

2. *Delirium.* The delirium may be mild. This is usually the case if the typhoid state or a subnormal temperature prevails. It is sometimes attended by delusions. There is subsultus often, and picking at the bed-clothing. The delirium may amount to true mania, and may be active and the patient exhibit other maniacal symptoms. On the other hand, the patient may be noisy, restless and sleepless. Melancholia and delusive insanity may develop after the violent nervous symptoms of uræmia pass off.

3. *Convulsions.* A convulsion may be the first indication of disease of the kidneys, or it may succeed a few days of persistent headache, or follow an attack of uræmic vomiting. The convulsion resembles epilepsy, and hence is known as an epileptiform convulsion. If recurring in rapid succession the interval is occupied by delirium or coma. If infrequent, the patient's mind may be clear in the intervals. Sometimes a focal or Jacksonian epilepsy occurs instead of the true epileptiform convulsion. The *temperature* is usually elevated. In worn-out subjects, or those who have had exhaustive diarrhœa or other debilitating cause, the temperature may be subnormal. A temporary blindness often follows the convulsion (*uræmic amaurosis*). Uræmic deafness may occur.

4. *Coma.* After the convulsion the mind may be restored, or the patient lapse into stupor followed by complete coma. Coma may develop without convulsions, or immediately succeed a general convulsion. Headache or eye symptoms may precede the coma. The patient in some instances lapses into a typhoid state in which the tongue is heavily furred and the breath very offensive. Unless the coma is very profound there is usually some twitching of the muscles of the hands and face.

5. *Local Palsies.* Dercum was among the first to call attention to the occurrence of monoplegia or hæmiplegia in the course of nephritis, which is distinctly of renal origin. The cases resemble central cerebral disease. The nature of the palsy is inferred by the results of the examination of the urine and the condition of the heart and arteries. Palsy develops suddenly, or may occur after a convulsion.

6. *Cramps* in the muscles of the calves, particularly at night, are of common occurrence, and should always lead to an examination of the urine.

7. *Pruritus*, local or general, is another nervous symptom which may be of uræmic origin.

8. *Pain* in the upper abdomen, or particularly in the median line, is a frequent precursor of more severe uræmic symptoms. It is of uræmic origin itself. It may be seated in either of the upper quadrants, and from thence extend to the lower portion of the abdomen.

Uræmic Dyspnœa. Modifications of the breathing often accompany symptoms of uræmia. The dyspnœa may be constant. It may occur in paroxysms, or both types may alternate. A common type in the uræmia of chronic nephritis is the Cheyne-Stokes breathing. Paroxysmal dyspnœa usually occurs at night, and resembles asthma in every respect. Cheyne-Stokes breathing continues, even through the period of coma, although not necessarily associated with it (see page 282).

Gastro-Intestinal Symptoms of Uræmia. Several forms are seen. 1. *Loss of appetite* is common. It is attended with absolute distaste for food after a small portion is taken. 2. *Nausea*, which may be continuous, or more frequently is limited to the early morning. 3. *Vomiting* may be paroxysmal, occurring chiefly in the early morning, or it may be sudden in onset, uncontrollable, and continue until nervous symptoms of uræmia develop. Urea is found in the vomit. The matter ejected is profuse, of a low specific gravity and at first acid in

reaction. If chronic, it may become alkaline. The odor is often sufficient to cause its recognition. 4. *Constipation* is generally the rule in the course of chronic Bright's disease. 5. *Diarrhœa.* One of the manifestations of uræmia is the occurrence of sudden, profuse, serous purging. This may be so extreme as to cause collapse, or may usher in coma and convulsions. 6. *Hiccough,* although a muscular affection, is usually associated with gastric disturbances.

Acetonæmia. Acetonæmia is a toxæmia which develops in the terminal stages of diabetes. It is due to an accumulation of acetone in the blood. It is also called *diabetic coma.* It develops acutely. A sudden onset is attended by sharp pain in the stomach with nausea, and frequently the occurrence of vomiting. At the same time there is severe dyspnœa. The breathing is irregular, and of a panting character. The patient is required to sit up in bed on account of the air-hunger. Restlessness begins at once. Delirium develops within the first hour. In a few hours coma sets in. The temperature is subnormal; the pulse irregular, and soon becomes weak and thready. The odor of acetone is detected on the breath.

Cardio-Vascular Symptoms. The symptoms are the effects of the retention of morbid products. First, the *heart and bloodvessels.* The poison which is not excreted circulates throughout the system. One of its effects is irritation of the vasomotor nerves of the bloodvessels. Excitation of these nerves causes peripheral contraction of the smaller vessels. At once obstruction to the flow of blood is created, so that, on account of the contraction, hypertrophy of the heart rapidly ensues. The first prominent symptom, therefore, is due to changes in the heart muscle.

Hypertrophy of the Heart. Of these the most pronounced is *hypertrophy.* The persistent spasm of the peripheral vessels causes increased arterial tension. The blood-pressure is raised and causes increased accentuation of the aortic second sound. *High tension* in the artery is recognized by the peculiar character of the pulse and by means of the sphygmograph.

Dilatation of the Heart. Unfortunately, it is not always that hypertrophy of the heart can be kept up. If it fails, we then have a second condition of the heart which is frequently found in renal inflammations; it is dilatation. This condition of the heart muscle is predisposed to by the state of the coronary arteries. The previously mentioned arterial tension favors the development of chronic endarteritis with general atheroma. The coronary arteries take part in this process. The endarteritis hinders cardiac nutrition, dilatation of the heart muscle follows, and later comes the development of two other conditions, atrophy and myocarditis. The above conditions are secondary to renal disease.

Here may be mentioned other relations of the heart and kidneys. *a.* We have renal disease following forms of cardiac disease. In dilatation of the heart passive congestion of the particular organ takes place. The kidney very quickly becomes the seat of such congestion. In the course of simple dilatation, or of valvular heart disease, the secondary

dilatation, passive congestion, and chronic inflammation develop slowly. Embolic process may also occur. *b*. Renal disease and cardiac disease may develop at the same time out of a common cause, as alcoholism, gout, or endarteritis.

In addition to high arterial tension and accentuation of the aortic second sound, the objective symptoms of atheroma of the aorta and arteries is present with the chronic inflammations of the kidney. These vascular changes need not be again rehearsed. (See Endarteritis.)

It is important, however, to bear in mind the proposition which indicates their frequent association, and also that in all instances of arterial disease the condition of the urine must be inquired into. It need not be said that symptoms due to rupture of the bloodvessels, particularly in the brain, or to aneurism, may necessarily be present in the course of renal inflammation.

Hemorrhages. The arteries are very liable to rupture, causing epistaxis, retinal hemorrhage, hemorrhages from the bowels and lungs, and hemorrhages underneath the skin. Frequent hemorrhages in large amount from any portion of the body should call attention to the condition of the urine.

Ophthalmoscopic Changes. Examination of the eye-ground should always be made, although attention is often directed to the eye by the complaints of the patient. The changes may occur in the acute or chronic forms of nephritis, although they are more common in the latter. 1. A diffuse slight opacity and swelling of the retina, due to œdema. 2. White spots or patches of various sizes, for the most part the result of degenerative processes. 3. Hemorrhages. 4. Inflammation of the intra-ocular end of the optic nerve. 5. Atrophy of the retina and nerve may sometimes result from and succeed the inflammatory changes. These changes may affect one eye only (Gowers). It must not be forgotten that temporary blindness may occur independent of retinitis.

Dropsy. Dropsy may occur in all forms of nephritis. It is most common in acute varieties, but it is also present in chronic diffused nephritis with exudation. Renal dropsy usually begins in the face. It may develop suddenly in acute forms. In the marked forms œdema of the eyelids may continue for a long time. From local œdema all variations may be found to the point of extreme anasarca. The serous cavities are often filled. The œdema is usually associated with a diminished amount of urine. Its improvement is attended by increased diuresis. Dropsy, in chronic disease, is usually due to dilatation of the heart (see page 92).

The Cutaneous Symptoms, and Appearance of the Face. In inflammatory affections of the kidney the appearance of the skin and expression of the face are often characteristic, and point at once to an examination of the urine. The face is pallid, and of an ivory whiteness. In the chronic forms the pallor gives way to an ashen-gray or a sallow complexion. In chronic nephritis the skin becomes dry and harsh, and rarely is covered with a powdery substance, giving it the appearance of frost on the skin. The powdery substance is due to urea.

Petechiæ. In the later stages of chronic inflammatory affections *hemorrhages* under the skin and in the mucous membranes are seen.

Anæmia. Anæmia is a frequent symptom in all forms of nephritis; it is usually marked. It is associated with the peculiar pallor just described and attended by all the symptoms of anæmia. The recognition of the anæmia is only possible by examination of the urine and the blood.

General Symptoms. The cause of renal disease, as far as symptoms pointing to the kidneys are concerned, is often latent. Instead of renal symptoms, a generally depraved state of the system is seen, with *emaciation* and *weakness.* Lassitude without cause demands an examination of the urine.

Respiratory Symptoms. In addition to uræmic respiratory phenomena, the occurrence of pulmonary complications may be the first indication that the condition of the urine should be inquired into. Bronchitis, pneumonia, and pleurisy are common complications.

Gastro-Intestinal Symptoms. Uræmic symptoms have been referred to. Fermentative dyspepsia, gastralgia, and constipation are of common occurrence.

Congestions of the Kidney.

Congestions of the kidney are acute and chronic, and depend upon changes in the circulation, whereby blood accumulates in the kidney.

Acute Congestion of the kidney is caused by the action of irritant poisons; follows surgical operations, particularly if prolonged, and may follow extirpation of one kidney. Kidneys that are the seat of disease are liable to become the seat of active congestion.

Symptoms. The urine is diminished in amount, or may be suppressed entirely. Only a small amount is passed at frequent intervals, or it can be secured by the catheter alone. Albumin is present in considerable amount, and blood and epithelial casts are numerous. Death may take place with symptoms of uræmia.

Chronic Congestion of the Kidney. It is also called passive congestion. This form of congestion is usually a part of general venous stasis due to disease of the heart or lungs, as valvular disease of the heart with secondary dilatation or pulmonary emphysema. It is quite common.

Symptoms. The *urine* is diminished in amount; dark in color; of high specific gravity, ranging from 1020 to 1030. Uric acid and urates are increased. Urea to the amount of from 10 to 12 grains to the ounce is passed in twenty-four hours. At first there is no further change, but later, albumin appears in small amounts in an intermittent manner. Later it is constant and increased in amount. Hyaline casts are found in the urine, and a few red blood-cells.

The condition is recognized by its association with congestion in other organs; by the diminution in the amount of urine, its high specific gravity, and excess of uric acid and urates. This form of congestion is serious, because it leads to chronic nephritis. The presence of the latter is recognized by the usual changes in the urine.

Inflammations of the Kidney.

The inflammations of the kidney are divided in accordance with the activity of the process and the degree of exudation or cell-proliferation that attends the inflammation. We, therefore, have the following varieties:

Acute exudative nephritis (acute Bright's disease).

Acute productive or diffuse nephritis (acute Bright's disease).

Chronic productive or diffuse nephritis with exudation (chronic tubular nephritis).

Chronic productive or diffuse nephritis without exudation (chronic interstitial nephritis).

Suppurative nephritis.

Tubercular nephritis.

Acute Exudative Nephritis or Glomerulo-Nephritis. In this form of nephritis there is congestion, exudation of plasma, transudation of red and white blood-cells, and changes in the epithelium.

Causes. It may occur without definite cause, save exposure to cold, and at times even without such history. It occurs in most of the infectious diseases. It is of common occurrence after scarlet fever, and in the course of pregnancy, and in septicæmia. It occurs in diphtheria, erysipelas, and pneumonia frequently. It is the expression of a peculiar type of typhoid fever. It may complicate dysentery and acute tuberculosis. It forms one of the modes of termination of diabetes.

Symptoms. The course of the disease may be mild, presenting only changes in the urine, or there may be in addition to decided changes in the character of the urine, local and general symptoms.

In mild cases the *urine* is diminished in amount; micturition is frequent; the color of the urine is increased, and the specific gravity is usually high. A small amount of albumin is found, and a few epithelial and blood casts, and sometimes blood. At the termination of the disease the casts are hyaline.

In severe cases the disease is ushered in by *chill*, attended and followed by *pain* in the loins, with *fever*, *headache*, and much restlessness.

The *urine* may be passed more frequently than usual, but in small amounts; or micturition may diminish in frequency or cease entirely. Examination of the urine reveals the characteristic changes. The quantity of the urine is lessened; the specific gravity is normal or increased. There is a large amount of albumin, and an abundance of hyaline, granular, epithelial, and blood casts. Free white and red blood-cells, and epithelium from the pelvis and tubules are found.

The fever continues; the pain in the loins is sometimes very severe, and may be taken for lumbago, unless an examination of the urine is made. Within the first forty-eight hours the characteristic symptoms that follow the chill and that attend the urinary changes are *headache*, *sleeplessness*, more or less *stupor*, muscular *twitchings*, or general *convulsions*. *Eye symptoms* may be complained of. Instead of cerebral symptoms, *dyspnœa* may be marked. With both, *nausea* and *vomiting* are of common occurrence. The *heart's* action is increased in force and frequency. The left ventricle rapidly becomes hypertrophied. The

aortic second sound is accentuated. The *pulse* is hard and exhibits the characteristic features of high tension. From the onset of the first symptom, or within the first week, two other striking phenomena arise. They are, first, the occurrence of *dropsy;* second, the occurrence of *anæmia.*

Dropsy or œdema is one of the most constant symptoms. It appears first in the face, especially the eyelids. It may be limited to this region. It is worse in the mornings. From the face, in bad cases, it extends to the lower extremities and to the scrotum, and from thence all over the body. Anasarca is the name applied to the general dropsy; the connective tissue is infiltrated with serum. It is recognized by the pallor of the swollen surface; the pitting on pressure; the absence of heat and of pain. (See page 92.)

Effusion may take place into the serous cavities, either the pleura, pericardium, or peritoneum, causing the symptoms due to effusion. In some instances there is œdema of the mucous membranes, as the conjunctiva, the soft palate, and the glottis.

Dyspnœa may be a pronounced symptom, due either to uræmia (uræmic asthma) or œdema of the glottis, effusions into the pleura, or to bronchitis. If dilatation of the heart occurs, dyspnœa may arise, due to that or to the secondary œdema of the lungs.

With or without the occurrence of nausea or vomiting there is always loss of *appetite*, and usually *constipation.*

The *fever* is usually moderate and irregular in type. *Prostration* is common; often there is emaciation. Symptoms of *uræmia* may occur at any time.

Exudative nephritis with excessive *pus* is of sudden onset, characterized by high fever and extreme prostration. There is rapid *emaciation* and the early development of the *typhoid state.* This is preceded by delirium, headache, and stupor, with great restlessness. There is but little, if any, dropsy. Large numbers of red and white blood-cells and the usual casts are found in the urine. There is not as much diminution in the urine as is usually seen. The disease may arise without cause, or complicate scarlet fever or diphtheria.

This form is very fatal, and resembles *acute meningitis,* from which it is diagnosticated by the change in the urine.

Acute Productive or Diffused Nephritis. In this form there is an overgrowth of connective tissue and excessive growth of the capsule cells in the glomeruli in addition to the lesions of the first form. The whole kidney is not necessarily affected, but only portions at a time. *Symptoms:* The onset is sudden. The subjective symptoms previously described are present in a marked degree. *Nervous* symptoms (uræmia) are most pronounced. *Dropsy* develops rapidly and to an extreme degree. There is rapid development of *anæmia* and *loss of flesh.* The remaining symptoms tally with those of the first affection.

The *urine* is scanty, bloody, and of high specific gravity. The microscopical appearances are like those of acute exudative nephritis. If convalescence is established the urine becomes more abundant, with a corresponding fall in the specific gravity. The albumin and casts may appear for a time, but eventually disappear.

Diagnosis. The diagnosis of acute nephritis of either form is based upon the examination of the urine. Ætiological associations are of value. The more pronounced cases follow scarlet fever or pregnancy. In the latter condition it usually advances slowly. There may be no symptoms until the occurrence of uræmia or acute lung symptoms. In some instances the disease resembles typhoid fever. In cases in which the onset is sudden with early uræmic symptoms it must not be mistaken for epilepsy, delirium, or mania.

Chronic Productive or Diffused Nephritis with Exudation. In chronic inflammations the formation of new tissue always takes place. They are divided, therefore, in accordance with the exudation. The exudation is from the vessels. *Causes:* This form usually follows acute productive nephritis and chronic congestions or degenerations of the kidney. It develops in the course of syphilis, tuberculosis, endocarditis, disease of the bones, and prolonged suppuration. Frequent exposure to cold and wet, a residence in damp dwellings, and the alcoholic habit are causal conditions. It usually occurs in middle life, more frequently in men. When it occurs as a primary disease it is usually found in young adults. *Symptoms:* The disease develops slowly. General symptoms may first be observed. *Dropsy* may develop at first and continue throughout the disease, or recur at long intervals. The appearance of the patient is striking. The skin is of a peculiar *pallor* and pasty in appearance. The sclerotics are very white. The *anæmia* which gives rise to the pallor may be very profound and often be typical of that seen in the pernicious form of anæmia. The anæmia is due to diminution in the hæmoglobin and reduction in the number of red blood-cells.

Headache and *sleeplessness* are common symptoms. Pronounced acute uræmia does not often occur. Chronic *uræmia* may prove fatal by the patients lapsing into a typhoid state in which delirium alternates with stupor.

The *urine* is variable in quantity and character. It must not be forgotten that the course of the disease and the urinary symptoms are often quite variable in chronic nephritis. The urine may be normal in amount, but during the exacerbations it is scanty or suppressed. The specific gravity and the amount of urea lessen. In the most rapid cases it varies between 1012 and 1020. In chronic cases it falls between 1001 and 1005. In the latter stages the amount of the urine and the specific gravity may both be increased. Albumin is present in large amounts. When the disease is most active, and the dropsy at its height, the quantity of albumin is very large. In the quiescent period of the disease the amount is lessened. Casts are abundant, both epithelial, bloody, fatty, and granular.

Retinitis albuminurica is frequently developed in the course of the disease.

Dyspnœa is a common symptom. The dyspnœa may be due to any one of the many causes previously described which promote this symptom in the course of nephritis. It is frequently limited to sudden attacks which develop in the night or early morning. There is often some bronchial catarrh.

Nausea and *vomiting* are usual symptoms. The appetite is lost. *Hypertrophy* of the *left ventricle* takes place in all cases, except in those who had been previously weakened by other disease. The right ventricle is often hypertrophied also. The second aortic sound is accentuated, and the pulse is of high tension. Symptoms arise on account of the profound *anæmia*, such as *headache* and *vertigo*.

The disease is characterized in its course by remissions and exacerbations. During the exacerbations any one of the prominent symptoms that occur in renal inflammations may be present. Œdema is the one symptom which occurs most frequently, and is likely to continue the longest. The disease lasts from three months to three years, and may pass into the second variety of chronic inflammation.

Course of the Disease. Delafield has well outlined the course. The constant symptoms are anæmia, dropsy, and albuminuria. 1. The symptoms may be continuous and progressive in severity, death taking place at the end of one or two years, on account of dropsy or uræmia. 2. The symptoms may continue for several months, and the patient finally improve. Recurrent attacks take place, the symptoms being more severe with each attack. In the intervals of the attacks there is a small amount of albumin. 3. The patient may apparently recover, but the urine continues to be of low specific gravity, and contains some albumin. A fatal attack of uræmia, or an apoplexy, or the onset of an acute disease, may cause an exacerbation of the renal symptoms. 4. The symptoms in a mild degree may persist for years, the patient at the same time feeling comparatively well. 5. Spasmodic dyspnœa may be the first and only symptom for a long time.

Chronic Productive or Diffused Nephritis without Exudation. This is the form of nephritis which is also called *interstitial nephritis, granular kidney* or *cirrhosis of the kidney.*

The kidneys are diminished in size, the capsules adherent, and the surface roughened. There is an overgrowth of connective tissue with atrophy in the epithelium and the tubules, and dilatation of some of the tubes, forming cysts.

Causes. This form of nephritis follows chronic congestion of the kidney, and is also caused by alcohol, lead, gout, syphilis, malaria, and by chronic endarteritis. The latter condition, as well as cirrhosis of the liver and pulmonary emphysema, frequently develop hand-in-hand with the nephritis. This form of nephritis is notably prevalent in several generations of different families, so that an hereditary history is often readily obtained.

Symptoms. The onset of the disease usually occurs late in life, although well-defined cases may occur as early as the twenty-fifth year. The progress at first is very insidious, and the disease may have advanced to an extreme degree without the occurrence of a single symptom. Death, indeed, may be due to other causes, or a person in perfect health may suddenly manifest symptoms of uræmia, or be seized with apoplexy or other usual complication.

The *urine* is increased in amount, clear in color, and of low specific gravity. The albumin is small in amount, or may be absent. Repeated examinations conducted over a considerable period of time, may disclose

its presence. Hyaline casts are present in small numbers. In some examinations it may require a dozen or fifteen slides to be passed over before they are found. Sometimes there are a few red blood-cells. Rarely in the course of the disease the urine may be bloody at irregular periods, or actual hæmaturia may take place. With the exception of the state of the urine the only symptom present may be loss of flesh and strength. At the same time the skin becomes dry and harsh. Œdema, however, is not usually present unless there is dilatation of the heart. Special symptoms are due to uræmia, to changes in the heart and arteries, and to neuro-retinitis.

The Heart. The left ventricle hypertrophies. The aortic second sound is accentuated. The arterial pulse is of high tension. The arteries become more prominent, and present all the signs of endarteritis. In the later stages, as nutrition fails, dilatation of the heart takes place with regurgitation at the mitral valve, and the development of a train of symptoms due to these changes. Among others we find general malaise, palpitation of the heart, dyspnœa, œdema, and visceral congestions.

Uræmic Symptoms. These symptoms may occur at any time in the course of the disease. Headache is most common and constant. It may occur in the early morning only, or continue throughout the day. It may be continuous and cause sleeplessness. General neuralgic pains may be present instead of severe headache. Muscular twitchings or general convulsions may be other pronounced symptoms, or, instead, delirium which may be mild or violent, stupor, and coma may come on. These symptoms may occur suddenly, or develop very gradually. In acute uræmia the above-mentioned cerebral symptoms occur, and at the same time there is peripheral spasm of the arteries, causing high arterial tension with elevation of the temperature. The fever may rise to 103° or 104°, but is usually about 102°, and is irregularly continuous. After the patient lapses into deep coma, if the attack is fatal, the tension of the pulse is lost, and it is increased in frequency and diminished in strength. In chronic uræmia the cerebral symptoms develop gradually. The temperature is likely to be subnormal. The pulse is rapid and feeble.

Pulmonary symptoms due to uræmia are quite common. They may be the first expression of uræmia. This is seen in all forms of nephritis. The most marked symptom is dyspnœa, which is spasmodic and of short duration. The attacks may occur frequently, and are usually increased by exertion and aggravated by a recumbent posture. The shortness of breath may occur in the early morning hours, or may continue throughout the day.

Gastro-intestinal Symptoms. Catarrhal gastritis almost always complicates nephritis. In addition, gastric symptoms due to uræmia, and hence to deficient action of the kidney, ensue. The most common is the occurrence of morning nausea or of morning vomiting; the occurrence of spasmodic vomiting at irregular periods, or the occurrence of violent, acute vomiting which is followed in two or three days by other symptoms of uræmia. The patients are usually constipated. When the disease is complicated with cirrhosis of the liver intestinal catarrh is

common, and intestinal ulceration with consequent diarrhœa is frequently found. The onset of uræmia may be characterized by violent and profuse serous purging, which of itself may cause collapse and death.

Neuro-retinitis is a frequent complication of nephritis, and may advance more rapidly than other complications, so that dimness of vision, blindness, or other eye symptoms may cause the patient to consult an oculist before attention is called to the condition of the kidneys. The occurrence of this complication points at once to the necessity of an examination of the urine.

It is common in the course of an interstitial nephritis to have occur accidents due to the condition of the arteries that accompany this disease. On account of the atheroma, aided by the hypertrophied heart, rupture of the vessels frequently takes place. Apoplexy is, therefore, of common occurrence, and hemorrhage into other organs sometimes occurs.

There is always a tendency to chronic inflammations of the mucous membranes and to acute inflammations of serous membranes in the course of chronic diffused nephritis. It is necessary, therefore, when local inflammations of this character are present, to make thorough and repeated examinations of the urine, especially in patients over forty with a history of one of the causal factors previously mentioned as operative in the individual.

In addition to the pulmonary symptoms of uræmia, symptoms referable to the lungs are common from other causes. The symptoms may be due to an intercurrent bronchitis, pneumonia, or pleurisy. Chronic bronchitis or œdema of the lungs may be present on account of dilatation of the right heart. The chief pulmonary symptoms that point to these conditions are dyspnœa and cough.

Course of the Disease. Several forms of interstitial nephritis are observed. In the latent form the disease may have advanced to an extreme degree, and death takes place from an intercurrent disease or accident, no symptoms of renal disease having been present during life. On the other hand, palpitation of the heart may be the only symptom complained of, and the observer finds a hard pulse, general atheroma, and hypertrophy of the left ventricle with accentuation of the second sound. Apart from this they may enjoy very good health. Their danger lies in the occurrence of pneumonia or inflammation of a serous membrane. Often the local inflammatory symptoms are slight or masked by the symptoms of renal disease, which develop rapidly.

In another group of cases some special symptom only may be complained of. In some instances it may be gastric catarrh, in others eye symptoms alone may be present, while in others hemicrania or other forms of headache are observed. With the headache there is usually vomiting. Again, we may see constant neuralgias on the one hand, or persistent muscular rheumatism to be the only symptom. Nose-bleed is a symptom which may be the only indication of chronic nephritis, particularly if the epistaxis occurs frequently.

In other cases the course is not latent, but characterized by a series of attacks at varying intervals.

During the attacks the symptoms resemble the acute form of nephritis, with acute uræmia, the occurrence of dyspnœa and of loss of appetite, nausea and vomiting. The high tension of the arteries is worse at the time of the attacks. The urine contains albumin, and is of low specific gravity during the time of the attack; during the interval the albumin is found at irregular times.

Spasmodic dyspnœa is the first, and sometimes the only symptom for a long period of time. After a time the renal symptoms become pronounced, pointing to the true nature of the disease. The renal disease is often not suspected until after the patient has had an attack of apoplexy. The course of this form of nephritis is varied very much by the occurrence of complications, notably emphysema, endocarditis, or cirrhosis of the liver.

Suppurative Nephritis (Abscess of Kidney). Infectious matter is conveyed to the kidney either through the *blood*, as in pyæmia and ulcerative endocarditis (rarely dysentery and actinomycosis), or by the *ureters*, as when it follows pyelitis or cystitis. A wound may infect the kidney directly.

Symptoms. The symptoms are those of the primary disease, and the affection is usually only recognized post-mortem. Or the symptoms are merely those of suppuration. Pus is seen in the urine only on rupture of the abscess into the pelvis of the kidney.

Tubercular Nephritis. Fever, emaciation, anæmia, and prostration characterize the course of the disease. Tuberculosis is usually found elsewhere. There may be no other symptoms. Sometimes hydronephrosis is present. A tumor is often present. It may be in the loins, or may be in front, above, and a few inches to the right or left of the umbilicus. The *urine* is normal or contains pus and detritus or even tubercle bacilli. The testicles and bladder should be carefully examined.

The Degenerations.

Degeneration may be either acute or chronic. The process is always secondary, due to the action of inorganic poisons, as arsenic or phosphorus, or the poison of infectious disease, or is produced as the effect of chronic disease of the organs, or by disturbance of the circulation.

In *acute degeneration* of the kidneys the urine is unchanged, or its quantity is diminished. It contains a little albumin, or the albumin is present in large amounts with casts and blood corpuscles.

There may be no symptoms except changes in the urine, or symptoms of uræmia may develop at once. Dropsy and hypertrophy of the heart do not occur.

Chronic degenerations in the kidneys follow chronic congestion, or are produced by alcoholism or syphilis. They occur in the course of pulmonary phthisis, of chronic suppuration, and syphilis; they may develop in the course of gout or malarial cachexia. *Symptoms:* In the simpler forms there may be no clinical symptoms whatsoever. In others there is loss of flesh and strength, the development of anæmia, and in rare instances the development of the typhoid state.

The changes in the urine vary. It may be abundant, scanty, or sup-

pressed. The specific gravity is not changed, but albumin and casts are found.

Amyloid degeneration of the kidney is associated with similar degeneration in other organs. It occurs in the course of phthisis, of chronic suppurations, of syphilis, of chronic dysentery, and is thought to occur in the malarial cachexia or with gout. *Symptoms:* The degeneration may be present without clinical symptoms. If symptoms arise they are due to the anæmia and cachexia that attend the primary disease, and to the involvement of the other organs in the same process, as the liver, spleen, and intestines. Œdema may be present, although it is more frequently absent. Uræmia is of rare occurrence. In the uncomplicated degenerations there is no hypertrophy of the left ventricle, and albuminuric retinitis is a rare occurrence.

The Urine. It may be diminished, normal, or increased; it varies from time to time in the same case. It is usually very pale in color. The specific gravity is not constantly at one figure. Albumin is constant, and usually is in considerable amount. Hyaline casts and white blood-cells are always found. When other casts are present nephritis probably complicates the condition.

The diagnosis of amyloid disease is based upon the presence of the cause; changes in the urine; and signs of similar disease in the other organs.

Sarcoma and Carcinoma of the Kidney.

Either disease may be primary or secondary. Sarcoma may be congenital. The tumor may occur at any age, but is relatively common in young children. In older persons it is often preceded by calculus. *Symptoms:* In some instances there are no symptoms during life. In others the disease may advance considerably before it presents any signs. If symptoms are complained of they are usually limited to pain, the occurrence of hæmaturia, or the development of a tumor. The pain is dull and seated in the lumbar region. It may be neuralgic in character, and, indeed, there may be a true *sciatica* with paresis of the leg from pressure of the tumor. The *tumor* is firm; its surface smooth or nodulated. It may be felt in the loins, and in front, above the umbilicus, a few inches to the right or left of the median line. The descending colon lies in front of the tumor. The *hæmaturia* may be constant or intermittent. The clots of blood may cause renal colic.

The general symptoms are those of carcinoma. A marked rapidity of the pulse has been noted in several cases. In girls a premature development of hair on the pubes and in the axillæ, and pigmentation of the skin have been observed.

The tumor must be distinguished from tumors of the lymphatic glands, of the liver, of the spleen, and of the ovary. It must not be confounded with psoas abscesses and perinephritic abscesses, which cause a tumor behind.

Cystic Kidneys.

1. *Congenital.* The kidney consists of a mass of small cysts filled with clear fluid. It may interfere with the birth of the child on account of its large size.

2. *Acquired.* The cause is trauma and obstruction of the ureter. The symptoms are those of a renal tumor which fluctuates. The urine may be normal or hæmaturia may be present.

Horseshoe Kidney. There are usually no symptoms. The kidney can usually be felt through the abdomen if its walls are relaxed, or by bimanual examination.

Hydronephrosis.

Causes. It may be congenital. Obstruction of ureter by stone; pressure of tumor; twist, as in floating kidney; exudates.

Symptoms. In addition to the symptoms of the causal condition we have upon the development of hydronephrosis the presence of a *tumor*, arising in the region of the kidney and extending toward the middle line. Sometimes fluctuation can be detected; often not. Variations in size of the tumor may occur with changes in amount of urine passed. Puncture and the finding of a fluid with elements of urine in it are valuable means of diagnosis; but if the hydronephrosis is old this fails, as the fluid loses its urinary character and cannot be distinguished from that of an ovarian cyst. When on one side, the urine may be normal; when on both sides, it is diminished; anuria and uræmia may occur. If pyelitis is present the urine takes character from that.

Pain may or may not be present. Gastric symptoms are very common. Constipation or diarrhœa is seen. Hypertrophy of the left ventricle may occur, as in *chronic* nephritis.

Nephrolithiasis (Renal Calculus).

Renal calculi vary in size from "sand," through "gravel," to "stones." The latter may be from the size of a cherry to one large enough to fill the pelvis of the kidney. They consist usually of uric acid, and are hard, brownish-red or blackish, crystalline, and the larger ones in distinct layers. More rare are calculi of calcium oxalate, extremely hard and nodular. Some stones have alternate layers of the two salts; others are of phosphates, but usually the inside is of uric acid or calcium oxide, the phosphates having been deposited after the urine became alkaline. Very rare forms are of cystin, xanthin, indigo, etc.

Symptoms. When stones are very small (sand) there are no symptoms except, perhaps, occasional pain in the lumbar region. When larger they attempt to pass the ureter or irritate the pelvis and cause *renal colic.* The latter comes on suddenly, is very intense, radiates from the loin and right or left center of the abdomen down to the bladder, testicle, and thigh. Collapse occurs in severe cases. The urine may be lessened in amount, or suppressed if both sides are obstructed. It may contain blood and pus. The attack lasts from a few hours to several days. Between paroxysms there may be constant pain, and the urine contains pus, pelvic epithelium, often blood; at other times the urine is clear and normal. Pyelitis, pyelonephritis, and hydronephrosis may develop.

The stone usually develops in the pelvis of the kidney—not in the kidney itself. A stone may remain fixed in the pelvis and produce no

symptoms, or those of gastric disturbances, catarrh of the bladder, and pyelitis.

Diagnosis. It must be distinguished from lumbago, perinephritic abscess, hepatic colic, and gastralgia.

Pyelitis. Pyonephrosis.

Causes. Rarely primary; usually secondary. Severe infectious diseases (typhus, variola, diphtheria, pyæmia); toxic substances ingested (cantharides, etc.); chronic nephritis; inflammation of the bladder or ureter; strictures of the ureter or urethra; hypertrophy of the prostate; spinal palsies of the bladder; calculus; parasites; blood-clots.

Symptoms. The Urine. Pus in the urine with pelvic epithelium—although it is not safe to base a diagnosis on the presence of the latter; casts of the canals opening into the pelvis are more characteristic; epithelial casts, and casts containing micro-organisms. The urine is often increased, acid, and contains pus and albumin, rarely blood. *Pain* in the region of the kidney, often severe, is complained of, although it may be absent. When present, it is often of a tearing character. *Tumor.* A tumor is often present. It is most prominent in the loin or in the abdomen. In the latter the mass can be felt two inches to either side of the umbilicus, usually above the tranverse line.

Fever is irregular, remitting, or septic. If the *bladder* is healthy its symptoms fail to aid in diagnosis.

Perinephritic Abscess.

Causes. Trauma; abscess in the kidney; pyelitis (either simple, calculous, tubercular, cancerous, echinococcal); abscess in neighboring organs, as the liver or lungs; Pott's disease; actinomycosis; pelvic cellulitis; appendicitis. It also occurs as a primary disease in apparently healthy individuals or after infectious diseases.

Symptoms. The *secondary forms* have symptoms of the primary disease, and later swelling and pain in the renal region.

Primary form. Chills and fever, pain, difficulty in defæcation. The general condition suffers. Finally, in all cases there is the formation of a swelling in the lumbar region, at first hard; then œdema of the skin is found and fluctuation detected. The abscess may descend and point above Poupart's ligament. It may press upward and cause dyspnœa. Great tenderness and pain in the region of swelling may arise and the pain radiate to the leg. Irregular septic fever and chills appear. *Urine* is not generally changed unless some communication with the pelvis or ureter has formed. The patient lies on his back, turned toward the affected side. The knee and hip of this side are flexed and the thigh rotated outward. The affection may simulate coxitis and appendicitis.

Parasites.

1. *Echinococcus.* Comparatively rare. Usually there are no symptoms until a tumor is felt. Then pain gradually develops. The cyst

may open into the pelvis of the kidney, and cysts or scolices be discharged, with colic.

Pyelitis and cystitis may also develop.

Echinococcus cyst may inflame and lead to general pyæmia. Puncture of the discovered tumor is otherwise the only means of diagnosis.

Hydronephrosis and ovarian tumors must be distinguished. Puncture is necessary.

2. *Distoma Hæmatobium.* Common in Egypt and Abyssinia. Eggs collect in great masses in the urinary passages, and lead to inflammation, ulcers, stenosis, etc. Eggs found in the urine alone make the diagnosis possible.

3. *Strongylus Gigas.* Very rare. Symptoms of pyelitis. (The parasite is of the size of a ground-worm.)

4. *Filaria Sanguinis Hominis.* Causes chyluria. Embryos may be found in the urine.

Floating Kidney.

Floating kidney is usually seen in women after the age of forty, who have had considerable hard work and borne many children. Its occurrence is frequently preceded by a history of unusual lifting or strain, followed by tearing or dragging sensations in the abdomen. Pain may continue for several weeks after the injury, and then subside and the occurrence be forgotten, or subjective sensations may continue.

The symptoms that arise are due to the local dragging or pulling of the kidney on its mesonephron, or to reflex symptoms, or to pressure upon adjacent organs.

The pain that attends floating kidney is usually referred to the right or left of the median line; sometimes to the hypogastrium. It may be constant, dull, and aching in character. Paroxysms may arise in the course of the constant pain, or a paroxysm alone take place. The paroxysms continue for three or four days, during which time other subjective symptoms are more pronounced. Nausea may occur with the paroxysms, or be more or less constant. Sometimes vomiting takes place. In addition to pain a dragging sensation is experienced, and the patient may appreciate the presence of a tumor or lump in the abdomen, as well as its movability when she moves about. The reflex symptoms are chiefly referable to the nervous system. Emotional disturbance is observed when the organ is out of its capsule. Hysteria may be present. There is often great depression of spirits, and often hypochondriasis. From pressure jaundice may occur, and the intestine may be occluded.

The urinary symptoms are of interest. When the local pain and other symptoms are more pronounced the urine may be scanty. In one case it was reduced to sixteen ounces in twenty-four hours. At the same time that the urine is scanty, hydronephrosis will develop. It will be referred to again. As the kidney slips back into its bed the twisting of the ureter is relieved, and discharges of large amount of urine take place. Palpitation of the heart is a common reflex symptom.

Objective Symptoms. The abdominal walls are usually relaxed, and may or may not contain a large amount of fat. On palpation a tumor can be found to the right or left of the median line, freely movable and

alternating its position with change in position of the patient. If the tumor is situated on the right side, it may be in close proximity to the liver, or be felt opposite the umbilicus, or often in the iliac region. When near the liver, by careful palpation the fingers can be introduced between the border of the liver and the mass. Usually it does not move with respiration, but sometimes it is found to do so. On the left side it may be as high up as the margin of the ribs. It generally is felt in the mid-clavicular line a little above the level of the umbilicus. It is likewise movable. On palpation the tumor is found to be of the shape of the kidney, firm in character, and at times quite painful. The hilus of the kidney and the vessels going to it, can at times be felt. Palpation frequently causes nausea, and may excite an attack of palpitation of the heart, or pronounced nervous symptoms.

In a case recently under the writer's care, about once a month the woman, aged fifty-five, would experience pain in the abdomen, to the right and above the umbilicus. At times nausea and vomiting accompanied the attacks. At other times marked depression or hysteria. Anuria always occurred and continued for a variable time, not longer than five days. With one of the paroxysms the tumor was found in the region of the gall-bladder, movable with respiration, but distinctly defined from the liver by placing the fingers between the lobe and kidney. It moved with each change of position of the patient, and at first the hilus could be distinctly felt. As the pain continued the anuria persisted, and a marked change in the tumor was observable. It gradually increased in size, and a portion of it fluctuated ; it was round and partook of the character of a cyst. The fluctuation was detected by placing the hand on the tumor in front and pressing firmly toward the other hand placed in the loin above the pelvis. When the anuria disappeared, a copious discharge of urine took place and the swelling subsided.

Floating kidney may be confounded with tumor of the gall-bladder, tumor of the pylorus, and with tumors in the pelvis. It is not likely to be confounded with an omental tumor, carcinoma, or tuberculosis, because the phenomena of these processes are not present and ascites does not occur, nor is there rise of temperature, as in many cases of tuberculosis. Tumor of the gall-bladder is distinguished by the absence of previous history or of symptoms or signs indicating disease of the gall-ducts. If jaundice is present, it is not so intense as in tumors of the gall-bladder. While the gall-bladder is movable, it is not so distinctly so as floating kidney. Anuria does not occur.

In cancer of the pylorus the emaciation and anæmia are more pronounced than in floating kidney. The vomiting, usually characteristic in that affection, and the physical signs of dilated stomach, can be made out. Tumors of the pelvic organs are determined by examination according to the usual methods.

CHAPTER VIII.

DISEASES OF THE BLOOD AND DUCTLESS GLANDS.

Inspection of the Blood.

THE blood consists of corpuscles and serum. The corpuscles are four: (1) red blood-cells; (2) nucleated red blood-cells; (3) blood-plaques; (4) leucocytes.

The ordinary red blood-cells measure $\frac{1}{3200}$ inch; the leucocytes, $\frac{1}{2500}$ inch. There are from 8000 to 15,000 leucocytes in a cubic millimetre of blood, or 1 to 350–700 red blood-cells.

Inspection of the blood may be (1) with the eye alone, or (2) with special instruments.

1. INSPECTION WITH THE UNAIDED EYE. This gives but little information. It serves to distinguish bright-red arterial blood from darker venous blood, and also indicates when arterial blood has become deficient in oxygen from any of the causes of venous engorgement and cyanosis. In chlorosis and hydræmias the blood is pale, as though mixed with water, while in severe leukæmias it has a slight milky tinge. On the other hand, in carbonic oxide poisoning the blood becomes of a brighter red, while in poisoning with chlorate of potash and anilin, and in grave cases of poisoning with nitrobenzol and hydrocyanic acid, it is brownish-red or chocolate-colored.

2. INSPECTION WITH SPECIAL INSTRUMENTS. These are the microscope, the hæmoglobinometer, the hæmocytometer.

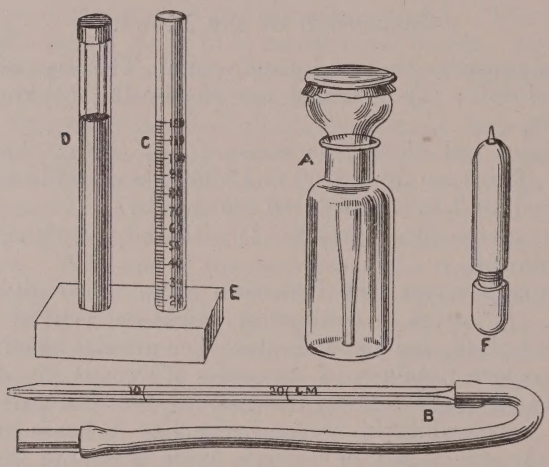
The Microscope. Inspection with the *microscope* reveals red and white blood-cells and blood-plaques. In an adult man the red cells number from 5,000,000 to 5,500,000 to the cubic millimetre; in an adult woman the number is usually less, being from 4,500,000 to 5,000,000. The proportion of white to red cells is about 1 to 500. There is a normal increase of white cells during digestion.

The microscope is, of course, essential to blood-counting, to the study of cover-glass preparations according to Ehrlich's methods, and to examinations for parasites.

Hæmoglobinometers. Gowers' hæmoglobinometer (Fig. 123) consists of (1) a closed tube, D, containing coloring matter representing the color human blood should have normally if diluted one hundred times; (2) a corresponding empty tube, C, graduated in an ascending scale from 10 to 120 per cent.; (3) a capillary glass tube, B, marked at 20 cubic millimetres; a small guarded lancet, F, and a small bottle with pipette-stopper, A, for distilled water. A few drops of distilled water are first placed in the empty tube, C, to prevent the coagulation of the blood, which would occur if the blood were first put in the tube. The finger or lobe of the ear, previously cleansed with water and

ether, is then deeply stabbed with the lancet, so that the blood will flow freely, care being taken to avoid squeezing the punctured part; 20 cubic millimetres of blood are then quickly drawn up in the capillary tube and at once blown into the graduated tube, which is shaken to allow the blood to become diffused in the water. The tubes containing the standard coloring matter and the diluted blood are now held up, side by side, against a sheet of paper, and more distilled water added, drop by

FIG. 123.



Gowers' hæmoglobinometer.

drop, with repeated shakings, until the colors in the two tubes match. The height to which the column of diluted blood and water has risen in the graduated tube represents the percentage of hæmoglobin contained in the blood tested.

Fleischl's hæmometer consists of a small metal table with an aperture in the middle, and under this a reflector made of plaster-of-Paris. The opening is occupied by a small well having a glass bottom and divided into two equal compartments. The standard color of the blood at different dilutions is represented by a wedge of glass colored with Cassius purple, which is of course pale in color at the extreme edge and deepens in intensity with its thickness. This wedge of glass is moved under the table by a rack and pinion, and is accompanied by a graduated scale. One-half of the well receives simply the white light from the plaster-of-Paris reflector, while the other rests upon the ruby glass and obtains light through it. A small pipette and several capillary tubes about $\frac{3}{8}$ inch in length and mounted on slender metal handles are employed to obtain the necessary amount of blood; each one of them will hold enough normal blood when properly diluted to produce a color corresponding to that of the ruby glass at the 100 mark. For use, one end of a capillary tube is carefully lowered upon a drop of blood which immediately fills it; the tube is then at once washed in one of the compartments of the well, which contains some water. The com-

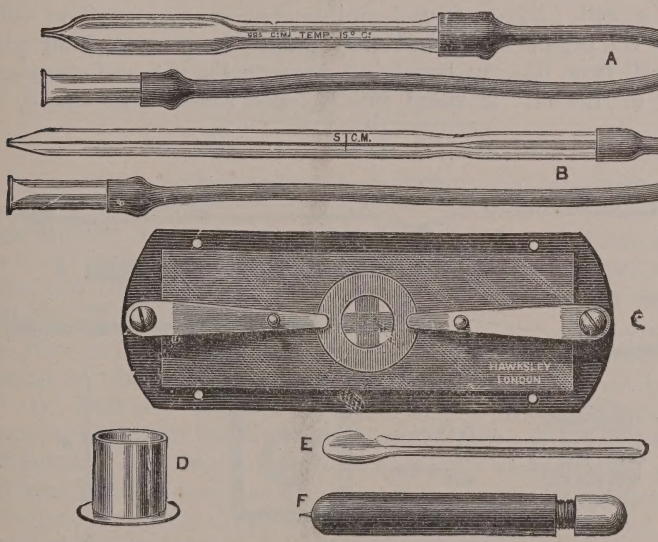
partments are now equally filled with water and the well so placed that the side containing blood receives white light while the other receives light through the wedge of glass. The glass is now moved by the rack and pinion until the intensity of the color in the two compartments is the same, and the percentage is then read off through the small opening behind the well.

These instruments are about equally accurate, and both are graduated for a higher percentage of hæmoglobin than is the average with Americans.

Hæmocytometers. The hæmocytometers, or blood-counters, most frequently used in this country are those of Gowers and Thoma-Zeiss.

Gowers' instrument consists (1) of a small pipette, A, which, when filled, holds exactly 995 cubic millimetres. It is for measuring the diluting fluid; (2) a capillary tube, B, graduated for 5 cubic millimetres; (3) a small glass jar, D, in which the dilution is made; (4) a small glass

FIG. 124.



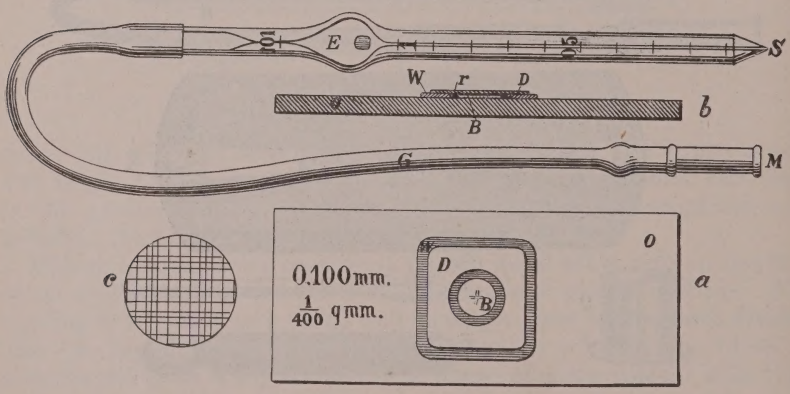
Hæmocytometer of Gowers.

stirrer, E, for mixing the blood and diluting fluid in the jar; (5) a small lancet, F; (6) a brass stage-plate, C, carrying a glass slip on which is a cell one-fifth of a millimetre deep. The bottom of the cell is divided into one-tenth millimetre squares. On the top of the cell rests the cover-glass, which is kept in place by the pressure of two springs proceeding from the ends of the stage-plate; 995 cubic millimetres of the diluting fluid are measured and blown into the mixing jar; then 5 cubic millimetres of blood are added and the two thoroughly mixed. A small drop of the mixture is then placed upon the cell, the cover-glass gently adjusted and held in place by the two springs. From five to ten minutes should be allowed to elapse, so that the corpuscles will have time

to settle to the bottom of the cell. The stage-plate is then placed under a microscope, and the number of red blood-cells in ten squares counted. This number, multiplied by 10,000, gives the number in a cubic millimetre of pure blood. It is better to count a large number of squares, take the average, and multiply by 100,000. This number is the product of the dilution (200) by the square surface of the cells, 100 (10×10), and again by 5, the depth of the cell: $200 \times 100 \times 5 = 100{,}000$. To facilitate seeing the fine lines marking the squares, a soft black lead-pencil should be gently rubbed over them before the drop of diluted blood is placed on the cell. Counting of the white cells is made much easier if the diluting fluid is colored a pale violet with a very small quantity of gentian-violet. The white cells then appear a distinct blue, while the red cells are unaltered. As diluting fluids, a 1 per cent. solution of common salt, or a $2\frac{1}{2}$ per cent. solution of bichromate of potash, as recommended by Daland, may be used.

Another hæmocytometer is the Thoma-Zeiss. It is preferred by most clinicians. It consists of a heavy glass slip (*a*) in the middle of which is a cell (*B*) exactly $\frac{1}{10}$ millimetre in depth. The cell is limited at the periphery by a circular gutter to prevent fluid placed upon the cell from flowing beyond it between the slip and cover-glass. The floor of the cell is ruled into squares whose sides are $\frac{1}{20}$ mm.

FIG. 125.



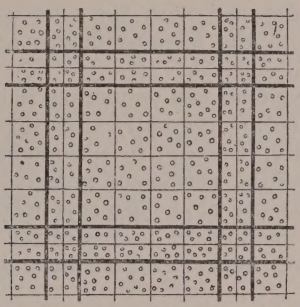
Thoma-Zeiss blood-counting apparatus.

Double lines mark out large squares containing twenty-five small squares. Thick, carefully ground cover-glasses (*D*) are provided in the case. The ordinary Potain *melangeur* (*S*) is used to measure and mix the blood. It consists of a capillary tube the upper portion of which is blown into a chamber (*E*) holding 100 c.mm. The stem of the tube is graduated at 0.5 and at 1 c.mm.

To use the instrument a drop of blood is obtained from the finger or lobe of the ear, and 0.5 or 1 c.mm. measured. The point of the tube is wiped quickly free from excess of blood, and inserted into the diluting fluid, which is drawn up to the level of the mark 101. The proportion of blood and diluting fluid is then $\frac{1}{2}$ or 1 to 100 c.mm.;

they are thoroughly mixed by shaking with the aid of a small glass ball contained in the chamber. The diluting fluid in the stem of the *melangeur* is now blown out, and a drop of the blood mixture placed on the cell. The cover-glass is now adjusted carefully to avoid bubbles, and to prevent the escape of the fluid between it and the slip. The cover-glass should now be pressed firmly down until Newton's color rings appear, and then the slip allowed to stand for five or ten minutes until the corpuscles have settled to the bottom of the cell. The number of corpuscles in twenty-five squares is now counted, and from this the number in a cubic millimetre of pure blood obtained by multiplying the

FIG. 126.



Appearance of blood in the Thoma-Zeiss cell.

average number in one small square by 400,000. This number is the product of 100 for the dilution, 400 (20 × 20) for the square surface of the cells, and 10 for the depth of the cells. The results are accurate in proportion to the number of cells counted.

The Hæmatokrit. The hæmatokrit is an instrument devised for the estimation of the number of red corpuscles by means of centrifugal force. In Daland's article[1] will be found a full description of the instrument, and from the same article the following method of using it is abstracted. The finger or ear and apparatus are prepared as above. An incision is made deep enough to produce a good-sized drop of blood. This is drawn up into a capillary pipette by means of suction through an attached rubber tube, and an equal amount of the diluting solution (2.5 per cent. solution of potassium bichromate) added and thoroughly mixed in a watch-glass. The hæmatokrit tube is then immediately filled by suction, one finger being placed over the free end when the rubber tube is removed, to prevent the loss of fluid. The filled tube is then placed in the frame of the hæmatokrit, and also a second prepared exactly as the first. The larger wheel is then rapidly rotated one hundred times, and the result read from the scale multiplied by 4[2] gives the percentage volume. (It was found by experimenting that each percentage volume represents about 100,000 corpuscles. We therefore add five ciphers to the percentage volume obtained, and the

[1] University Medical Magazine for November, 1891.
[2] 2 for the dilution and 2 to make it a percentage, as there are only 50 divisions on the scale.

number of red corpuscles per c.mm. is indicated.[1]) The whole procedure should be done as *quickly* as possible. Daland found the pipette made by Zeiss for measuring and diluting the blood in the estimation of *white* corpuscles to be particularly serviceable in diluting the blood for the hæmatokrit. The blood is drawn up to the 1 mark, a bubble of air is then admitted and the solution drawn up to the same mark, the blood ascending further into the pipette. They are then blown into a watch-glass and are so mixed ready for the hæmatokrit tubes.

Oligocythæmia. Oligocythæmia is the name applied to a diminution in the number of red blood-cells, from whatever cause. It is usually associated with *oligochromæmia* (deficiency of hæmoglobin), which, however, in idiopathic anæmia is absolute, not relative. Oligocythæmia, when marked, can be detected with the microscope alone, and can be estimated accurately with the hæmocytometer or hæmatokrit (see Fig. 127).

Leucocytosis. Leucocytosis is a temporary increase in the number of white blood-cells. It occurs normally after digestion and in newborn children. Pathologically, it occurs in pneumonia, in glandular swellings, sarcoma, osteomyelitis, pernicious anæmia, and chlorosis. It is best determined by the use of a hæmocytometer. Dry preparations according to Ehrlich's method are necessary for a study of the various forms of leucocytes (see under Leucocythæmia, page 693, and Figs. 128 and 129).

Poikilocytosis. This is a condition in which the red blood-cells are very irregular in shape—oval, pointed, angular, or reniform (see Fig. 127). It is a common accompaniment of severe anæmia, particularly leucocythæmia and idiopathic anæmia.

Microcythæmia. This is a condition of the blood characterized by the presence of cells containing hæmoglobin, but much smaller than an ordinary red corpuscle. They are found in anæmias and toxæmias.

Melanæmia. Melanæmia is a rare condition in which black, brown, or yellow granules are seen floating, either free among the blood-cells, or, more commonly, enclosed in cells resembling leucocytes. They are present in malarial fevers, particularly the chronic forms, and in relapsing fever.

Anæmia.

Anæmia is a condition characterized by a reduction in the number of red blood-cells, or of their contained hæmoglobin, or of the albumin, or of all combined.

For clinical purposes it is convenient to make a number of divisions of anæmia, though on ætiological and pathological grounds a number of them will no doubt soon be grouped together.

The following classification of anæmias, modified from Griffith,[2] is helpful in the study of anæmia. In it both pernicious anæmia and chlorosis are regarded as hæmolytic in origin, the destructive agent probably being absorbed from the intestine.

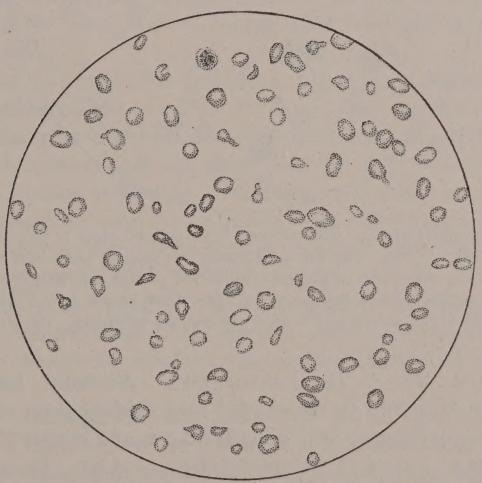
[1] Thus if the scale shows 12, we multiply by 4 to get the percentage volume, and add five ciphers, $4 \times 12 = 48$, = 4,800,000.

[2] "Diseases of the Blood," Keating's Cyclopædia of the Diseases of Children, 1890, iii. 770.

- Anæmia,
  - Non-cytogenic,
    - Hæmolytic,
      - Pernicious anæmia.
      - Other toxic anæmias.
      - Chlorosis.
      - Parasitic anæmia (some forms).
    - Oligocythæmic,
      - Parasitic anæmia (some forms).
      - Post-hemorrhagic anæmia.
      - Anæmia from loss of albumin.
      - Anæmia of malnutrition.
  - Cytogenic,
    - Leucocytic,
      - Leucocythæmia,
        - Splenic.
        - Lymphatic.
        - Medullary.
    - Non-leucocytic,
      - Splenic anæmia.
      - Lymphatic anæmia.
      - Hodgkin's disease.

I. Toxic Anæmias. Anæmia may be *toxic* in origin, the poison being developed either in the body or introduced from without. Toxæmia is at least sometimes a factor in the anæmias which develop in the course of acute infectious diseases or during convalescence from them; according to Hunter, pernicious anæmia would be classed under this head. The metallic poisons, particularly lead, mercury, arsenic, phosphorus, the potassium salts, especially the chlorate; certain of the antipyretics, notably pyrodin, and the aniline preparations are capable of producing anæmia.

Fig. 127.



Severe anæmia. (Reproduced from colored plate.) Dry preparation. Stained with eosin-methyl-blue. × 300. Great poikilocytosis of red cells. Many macrocytes and microcytes. To the left above, a mononuclear leucocyte with bluish nucleus and nearly unstained cell-body.

II. Parasitic Anæmias. Anæmia may be *parasitic*. 1. To this class belongs the anæmia of malaria, which is believed to be due to the *plasmodium malariæ* described by Laveran.

2. Certain intestinal worms are found associated with marked anæmias. (*a.*) The *bothriocephalus latus* sometimes produces a disease closely resembling pernicious anæmia, but whether by direct destruction of the

blood or by the development of toxic products themselves destructive, is uncertain; it may be present in large numbers without giving rise to anæmia.

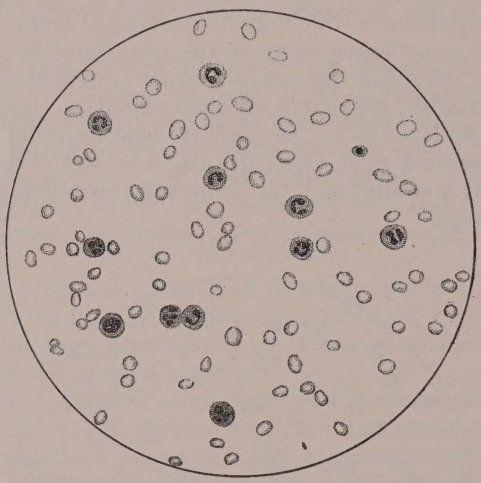
(*b*) The *ankylostomum duodenale* is believed to be the cause of the anæmia known variously as Egyptian or African chlorosis, tropical anæmia, brick-burner's anæmia, etc.

(*c*) The *anguillula intestinalis* is the cause of "Cochin-China diarrhœa" and its associated anæmia.

3. The *filaria sanguinis hominis* may produce anæmia by blocking up the lymph channels.

4. The *Bilharzia hæmatobia* may produce anæmia by inducing hæmaturia.

FIG. 128.



Grave anæmia with leucocytosis. (Reproduced from colored plate.) Dry preparation. Stained with eosin-methyl-blue. × 300. The red blood-cells are scanty, pale, and show poikilocytosis; the white cells, with the exception of one cell (to the left, below) are polynuclear, with dark-blue nucleus and faintly stained violet cell-body. To the right, above, is a nucleated red blood-cell (microblast) with dark-blue nucleus and faintly stained red cell-body. (H. RIEDER.)

III. ANÆMIA FROM HEMORRHAGE. Anæmia may be due to *hemorrhage*. In addition to accidental and *post-partum* causes, purpura, hæmophilia, menorrhagia, and metrorrhagia are frequent causes.

IV. ANÆMIA FROM CONSTITUTIONAL AND LOCAL DISEASES. Anæmia is often a marked symptom of *constitutional* and *local diseases*, such as tuberculosis, syphilis, cancer, rheumatism, scrofula, scurvy, rickets, Bright's disease, chronic catarrhal gastritis, and others. The anæmia here may be due to the malnutrition and interference with digestion brought about by the disease, or, as in the case of Bright's disease, in part to the direct loss of albumin, and in dyspeptic conditions to inability to take and assimilate food.

V. ANÆMIA OF MALNUTRITION. Anæmia may also be the result of malnutrition from deficient or improper food, or from the poisonous influences of unsanitary surroundings.

Chlorosis. Chlorosis is a form of anæmia occurring especially in young girls about the period of puberty, and characterized by great pallor of the skin and mucous membranes, with a greenish tint of the skin, a pearly eye, languor, weariness, suppression or irregularity of menstruation, venous hum in the vessels, dyspnœa, palpitation, dizziness, neuralgias, and an unstable condition of the nervous system. In spite of the extreme pallor there is usually but little loss of flesh. The bowels are usually constipated, the urine abundant, pale, and of low specific gravity. The digestion is disturbed, the appetite capricious, and the patients sometimes crave unwholesome things, such as earth, slate-pencils, vinegar, and the like. A systolic murmur over the base of the heart is common. Gastralgia is more common than in other forms of anæmia.

The changes in the blood are very important. There is always a marked reduction in the hæmoglobin, the percentage falling sometimes to 30 or 25 per cent. of the normal. The red blood-cells are usually also reduced, but not in the same proportion as the hæmoglobin. For example, there may be 4,000,000 red cells, but only 30 per cent. of hæmoglobin. Sometimes there is no diminution in the number of red cells; the latter, however, appear pale, vary considerably in size, microcytes and macrocytes and occasionally poikilocytes are present, and in severe cases nucleated red corpuscles are found. The number of leucocytes varies but little from the normal, but there may be a slight increase. Occasionally there is a rise of temperature, but it is probably due to some complication.

The cause of chlorosis has not been determined satisfactorily. Virchow has established the existence of congenital narrowing of the blood-vessels. Sir Andrew Clark thinks it is due to the absorption of poisonous matter from the intestine; the great benefit that follows saline purgatives in many cases indicates that fæcal toxæmia is a factor in these cases. Forchheimer[1] also looks upon it as intestinal in origin.

Sex and puberty are predisposing causes; but chlorosis may occur in boys, and appear in girls before puberty, and in young women considerably after that period. The prognosis is favorable; it may, however, be complicated with gastric ulcer, chorea, tuberculosis, and endocarditis. Recovery is often slow and interrupted by relapses.

Idiopathic Anæmia. Idiopathic, or pernicious, anæmia is a form in which the diminution of red blood-cells reaches an extreme degree. It occurs without adequate known cause, and runs, with remissions, a progressive course, and usually terminates in death.

The disease usually develops slowly and insidiously, the patient presenting the ordinary symptoms of anæmia—pallor, weakness, shortness of breath, palpitation, venous murmurs, loss of appetite, and impaired digestion. As the disease progresses the skin becomes of a pale lemon hue, weakness and dyspnœa increase, the patient has attacks of dizziness, faintness, and ringing in the ears; there may be slight œdema, and hemorrhages from the nose, the bowels, and into the retina,

[1] Trans. Assoc. Amer. Phys., 1893.

occur. The hemorrhages are small and distinct in the skin and mucous membranes. The urine is of low specific gravity and usually contains an increased amount of uric acid. According to Hunter, the urine should be dark and contain a large amount of pathological urobilin, some renal epithelium, a few casts containing blood pigment, and an increased amount of iron. The bowels may be disturbed by diarrhœa.

A peculiarity of the disease is the occurrence of fever of an irregular type. The temperature rarely rises higher than 102° or 103° in the evenings and is followed by a morning remission. It is not usually present in the early stages of disease, may be absent for weeks at a time when the disease is fully developed, and may cease entirely in the later stages.

In spite of the extreme exhaustion, anæmia, and widespread functional disturbance, there is no emaciation; the patient appears well nourished.

The blood appears pale and watery to the naked eye; there is difficulty in obtaining by puncture a sufficiently large drop for examination. The specific gravity is lowered, being 1028 instead of 1055. It has been found deficient in fibrin, iron, and nitrogen.

The blood changes in idiopathic anæmia are characteristic, and are essential to the diagnosis of the disease. In brief, they are: (1) very great reduction in the number of red blood-cells; (2) an absolute diminution in the amount of hæmoglobin, but, as compared with the number of red cells, a proportionate increase; (3) considerable variation in the size of the cells, the average size of the cells probably being larger; (4) poikilocytosis; (5) nucleated red blood-cells; (6) degenerative cells.

Reduction in the number of red blood-cells (oligocythæmia) reaches a more extreme degree in pernicious anæmia than in any other disease; the number often falls below 1,000,000, and in one case reported by Quincke[1] the number was only 143,000 per cubic millimetre. The shape of many of the cells is altered; they are oval, elongated, bent, or have projections of their substance (poikilocytosis). The size of the cells varies; there are microcytes and megaloblasts; but the occurrence of a distinct proportion of large nucleated red blood-cells (megaloblasts) is regarded by Ehrlich as almost diagnostic. The average size of the red cells seems to be increased, and so is the proportionate amount of hæmoglobin in each cell. The latter is a very characteristic symptom (the only one, according to Hunter). There are also red corpuscles which are stained by methylene-blue; these are regarded as degenerative by Ehrlich. The leucocytes are "usually diminished in number, showing a relative increase in the small mononuclear elements (lymphocytes, small transparent forms), while the multinuclear elements are relatively diminished, sometimes being under 50 per cent."[2]

The blood condition is not constant, but is subject to wide variations. Von Noorden has recently found that in a very short time a change in the form of the blood, a "formal" crisis, may occur, of such a character that before a period of improvement a "formal" overflow

[1] Deut. Arch. für klin. Med., Bd. xx.
[2] W. S. Thayer: Boston Med. and Surg. Journal, February 16 and 23, 1893.

of the blood with polynuclear leucocytes and nucleated red blood-cells occurs, whereas, before a period in which the blood becomes worse, and before the final stage, the blood becomes poor in leucocytes and in nucleated red blood-cells.[1]

The ætiology of the disease has not been determined satisfactorily. It is more common in Germany and Switzerland than in other parts of Europe or America. It occurs most frequently after the twentieth year, and between that and the age of fifty. Excluding the influence of pregnancy and parturition, sex makes no difference. Previous exhausting disease, chronic gastric and intestinal catarrh, great physical over-exertion, exposure, great shock or fright, precede in certain cases the development of the disease.

Petrone and Halst regard the disease as infectious and its germ identical with that found by Frankenhauser. Von Jaksch supposes that it is brought about by a living contagion. Hunter traces the cause to a poison produced by bacteria in the gastro-intestinal canal. The cases of Gibson,[2] in which cure or great improvement followed the use of beta-naphthol, tend to support Hunter's view.

There are no constant post-mortem lesions in pernicious anæmia, unless it be the deposit of iron in the peripheral zone of the liver cells.[3]

Diagnosis. The most important diagnostic features of the disease are extreme oligocythæmia, relatively high percentage of hæmoglobin, great poikilocytosis a noticeable number of large nucleated red blood-cells (gigantoblasts), and average increase in the size of the cells, and all this without emaciation or discoverable local disease which can bear a causative relation to the anæmia. In addition, retinal, subcutaneous and submucous hemorrhages, a urine with high specific gravity, high color, with urobilin in excess, alternating with urine of low specific gravity, in the absence of organic disease, point to *idiopathic* or *pernicious* anæmia.

Leucocythæmia.—Leucocythæmia, or leukæmia, is a chronic disease of the blood-making organs characterized by a great and persistent increase in the white blood-corpuscles; a diminished number of red blood-cells, which are altered in shape and size, and display nucleated and degenerate forms; a lessened amount of hæmoglobin, and by enlargement of the spleen, lymphatic glands, or medulla of bone. The disease occurs twice as frequently in men as in women, and two-thirds of the cases appear between the twentieth and fiftieth years. In women pregnancy, parturition, and the cessation of menstruation are causative factors, while in both sexes depressing influences upon body or mind, and antecedent disease, particularly malarial fever, have a distinct influence.

Gowers[4] believes that a history of intermittent fever can be traced in one-fourth of the 150 cases collected by him.

The first symptom noted is generally enlargement of the abdomen;

[1] Quoted by Weiss, Diagnostiches Lexikon.
[2] Edinburgh Medical Journal, Oct. (?), 1892.
[3] Hunter: Lancet, 1888.
[4] Reynolds' System of Medicine, Philadelphia, vol. iii. 481.

subsequently the patient complains of pain in the splenic region, weakness, dyspnœa, hemorrhage, œdema, and digestive derangements. Occasionally profuse hemorrhage from a trifling cause, as the drawing of a tooth, has been the earliest symptom noted. The increase of white cells and diminution of red cells is progressive, and soon makes itself evident in the pallor of skin and mucous membranes, and in increasing weakness and dyspnœa.

In the *splenic form* of the disease the spleen steadily enlarges, but may attain considerable size before the patient becomes aware of it. The enlargement is not usually painful, but gives rise to a feeling of distention, weight, and dragging. There may be tenderness on palpation and pressure, and sometimes the patient complains of sharp stabbing pains, due either to attacks of local peritonitis or to sudden enlargement of the spleen and consequent stretching of the capsule. The splenic enlargement is uniform, so that its shape and characteristic notch are unchanged. Moreover, the spleen remains in contact with the abdominal walls, lying in front of the splenic flexure of the colon, pushing aside the descending colon and small intestines, moving with respiration, and presenting the usual physical signs of a solid organ. Not infrequently the enlargement is so great as to fill the left hypochondriac and iliac regions, and reach beyond the middle line toward the right groin. Sometimes a venous hum can be heard over it.

As the result of this enlargement the diaphragm is pushed upward, increasing the dyspnœa already caused by anæmia, and sometimes inducing palpitation. The gastric functions are disturbed from pressure, vomiting, and other symptoms of dyspepsia being common.

A rise in temperature is a very common symptom. The fever is of irregular type, usually with nocturnal exacerbations, the temperature not often rising above 102°. The febrile type may be intermittent or remittent, and sometimes there are periods of apyrexia.

The pyrexia is said to be the most marked toward the close of the disease. Gowers states that the cases in which there is most fever are usually those of rapid course, considerable dropsy, and extensive hemorrhage.

As the disease progresses, weakness increases; anæmia becomes more intense; dropsy of the subcutaneous tissues, peritoneum, or pleura occurs; hemorrhages from the nose, gums, bowels, stomach, lungs, or uterus further exhaust the patient; digestion is poor, and diarrhœa is common.

Headache and tinnitus are frequent symptoms, occasionally delirium and coma may occur, and deafness is not uncommon toward the close of the disease. The eyes may be the seat of leukæmic retinitis.

The liver is enlarged, often to a considerable degree, but without special symptoms. The same is true of the lymphatic glands and other adenoid tissue. The marrow of the bones becomes the seat of disease in some cases, but it does not usually give rise to symptoms during life; certain bones, however, may be tender.

The Blood. The most characteristic and important changes from a diagnostic point of view occur in the blood. The blood when drawn from the finger is strikingly pale and whitish, an appearance supposed

at one time by Bennett to be due to admixture of pus. It coagulates slowly, is of lower specific gravity than normal, and its alkalinity is diminished. When placed under the microscope it is at once seen that the number of white cells is greatly increased. If a drop of blood be mixed with some distilled water containing a small quantity of gentian-violet, the white cells are stained a decided blue and can be picked out with the greatest ease. Instead of there being one white cell to 300 or 500 red, the ratio falls as low as 1 : 5, or 1 : 3, or even lower. Authorities differ as to the degree of increase necessary to distinguish leucocythæmia from leucocytosis, some including all in which the ratio is 1 : 50 or lower, and others excluding those in which the ratio is greater than 1 : 20 or 1 : 12.

Not only are the white cells greatly increased, but they vary considerably in size and react differently to staining fluids.

Ehrlich has described five varieties of leucocytes. The important points in regard to their presence are: (1) the small mononuclear elements are diminished; (2) the great difference in size of the multinuclear elements; (3) the presence of myelocytes, elements in which the protoplasm is filled with fine neutrophilic granules; (4) the presence of a normal proportion of eosinophiles in so extensive an increase of leucocytes.[1] Satisfactory study of these can be obtained only by cover-glass preparations. The greatest care should be taken to have a perfectly clean, dry cover-glass, which should be handled with forceps to avoid moisture and soiling. A small drop of blood is pressed between two cover-glasses, as in the preparation of sputum for staining. The blood may be then "fixed" by being heated at a high temperature for some time, or by immersion for half an hour in a solution of equal parts of absolute alcohol and ether. The prepared cover-glass should then be immersed for a few minutes in a solution of eosin:

Eosin	0.5
Alcohol (70 per cent.)	100.0

This solution should be diluted one-half before using. The cover-glass should then be dried and stained for three or four minutes in a saturated aqueous solution of methylene-blue, also diluted one-half before using. The red corpuscles are stained red, the nuclei blue, the eosinophile granules a brilliant red. Thayer says the following makes a satisfactory solution:

Saturated aqueous solution of acid fuchsin	2
Water	3
Saturated aqueous solution of orange-green	6.25
Saturated aqueous solution of methyl-green	6
To be added, drop by drop, while shaking the solution:	
Water	15
Alcohol	10
Glycerin	5

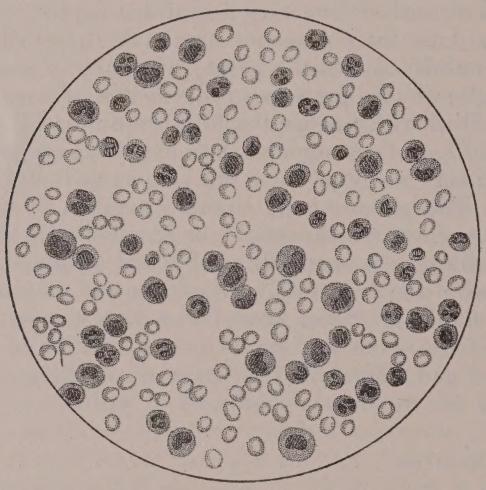
The specimen, fixed as before, is stained in this solution for from two to five minutes, washed in water, and dried in the air, or, if the specimen has been heated for an hour or more, between filter-paper, and

[1] W. S. Thayer: loc. cit.

mounted in oil or balsam. Specimens heated for one or two hours stain better than those which have been treated only a short time. The red cells appear orange or buff, the nuclei of the colorless corpuscles green, the neutrophilic granules a violet or lilac color, the eosinophilic granules a deep red. The nuclei of nucleated red corpuscles, when present, are stained an intense deep green, almost black.[1]

The essential points in the diagnosis of leucocythæmia are: 1. Such an excess of leucocytes in the blood that the ratio of white to red falls below 1 : 50 or 1 : 20; if the ratio is higher, the white cells should show a progressive increase. The individual leucocytes vary in size and other characteristics, as already described. 2. Enlargement of the spleen or lymphatic glands. 3. The occurrence of hemorrhages and

FIG. 129.



Mixed leukæmia. (Reproduced from colored plate.) Dry preparation. Fixed with picric acid. Stained with hæmatoxylin. × 300. The red blood-cells clear bluish-gray, two of them (one to the right of the middle, one to the left below) nucleated, with nucleus stained a deep dark-blue, almost black. The white cells for the most part mononucleated; several are polynucleated, of moderate size, with dark-blue nuclei. (H. RIEDER.)

dropsies unexplainable by disease of heart, kidneys, or other organs. 4. The symptoms of anæmia in a high degree, as dyspnœa. 5. Leukæmic retinitis. 6. Anæmic fever.

In *splenic anæmia* there is present the same enlargement and the general symptoms, though hemorrhage is not so common. Leucocythæmia is distinguished from it by the great excess of leucocytes and by their special characteristics.

In *lymphadenoma*, or *Hodgkin's disease*, there is extreme anæmia, though the excess of leucocytes present in leucocythæmia is seldom reached and the cells are smaller. The glandular enlargement of lymphadenoma is an early and constant symptom, the spleen not being much enlarged. The cervical glands are the ones usually first involved.

[1] Thayer: loc. cit.

The *duration* of leucocythæmia is usually two or three years; but some cases terminate in six months or even less, and some last six or seven years. The size of the spleen and the degree of oligocythæmia appear to have no influence. Gowers states that the cases in which enlargement of the lymphatic glands is an early symptom run a course apparently much more acute than others, but he admits that the number of such cases is comparatively small.

Death results most frequently from gradual loss of strength. Hemorrhage from various organs and surfaces is the immediate cause in many cases. It occurs in about three-fourths of the cases, and when not directly fatal increases the pre-existing asthenia. Diarrhœa and pulmonary complications are not infrequent causes of death.

Splenic Anæmia. Splenic anæmia is a disease of the blood-making apparatus characterized by enlargement of the spleen and progressive anæmia, the special features of which are decided diminution of the number of red cells without marked increase of the white.

The disease develops very gradually, and usually escapes observation until the spleen has attained considerable size. The anæmia appears to keep pace with the splenic enlargement, gradually increasing in intensity as the spleen enlarges. The clinical symptoms are those of anæmia—pallor, weakness, dyspnœa, palpitation. In the later stages of grave cases dropsies occur and hemorrhages, especially into the skin (petechiæ and ecchymoses) and from the nose, are liable to occur. The hue of the skin is that of yellow wax. There is not usually much loss of flesh, but the loss of muscular power is extreme. Fever of an irregular type is a common symptom. Mental dulness and drowsiness may be present. Appetite and digestion are impaired and the bowels irregular. The urine is free from albumin.

The red blood-cells are diminished in number, and may fall as low as 2,000,000 or 1,000,000. Nucleated cells are present and so are poikilocytes in severe cases. Leucocytosis is usually of moderate degree.

Hodgkin's Disease. Hodgkin's disease (pseudo-leukæmia, lymphadenoma, or lymphatic anæmia) is a disease characterized by enlargement of the lymphatic glands throughout the body and of other adenoid tissues also; by progressive oligocythæmia without, in most cases, much increase of the leucocytes; and by the development of lymphatic tumors in unusual situations.

The disease is most frequent in the first half of life, three-fourths of the cases being in males.

The first symptom noted is enlargement of the glands of the neck, sometimes of the inguinal, less frequently the axillary; rarely the tonsils are the first to be affected. The enlargement is painless and progressive, appearing first on one side of the neck and extending under the jaw to the opposite side. The tumors at first are distinct and movable under the skin. The swollen glands may remain in this condition indefinitely for months or years; but eventually they begin to enlarge very rapidly, lose their separate identity, and coalesce into large masses.

Other glands in remote parts, as the axilla and groin, retro-peritoneum, and arm, are affected. They may be soft and fluctuating or very dense and hard, but heat, tenderness, suppuration, and other evidences of inflammation are absent.

The spleen becomes enlarged, and may reach very great size, but rarely attains the dimensions common in leucocythæmia.

Other adenoid tissue in the intestine, tonsil, and posterior nares, and even the thymus, may enlarge and give rise to pressure symptoms.

Fever is a very constant symptom, but the type is not constant.

The onset of the disease may be marked by fever and constitutional symptoms, and the glandular enlargement appear later. On the other hand, in three cases reported by J. Dreschfeld,[1] all the patients enjoyed good health and were able to follow their work until a few weeks before death. In all, symptoms appeared suddenly with pain, weakness, pallor, loss of appetite, and pyrexia.

Coincident with the rapid and extensive enlargement of the glands, anæmia becomes pronounced, and is accompanied with the usual symptoms. Cough is often associated with the dyspnœa, and in women menstruation may cease.

In addition to the general symptoms there are numerous local ones due to pressure or impairment of function—cerebral anæmia from pressure on the carotids; cerebral congestion from pressure on the veins of the neck; disturbance of the heart from pressure on the pneumogastric; deafness; difficulty in deglutition and mastication, and pleural, peritoneal, and pericardial effusions.

The most frequent complications are nephritis, fatty degeneration of the heart, pleurisy, and less frequently pneumonia and pericarditis.

The *duration* of the disease is from six to eighteen months. Two-thirds of fifty fatal cases referred to by Gowers[2] ended in less than two years. It is difficult to determine accurately the beginning of the disease; sometimes a long period of latency follows the early glandular swelling; sometimes a general anæmia precedes any noticeable swelling of the glands, and sometimes the disease runs an acute course, ending fatally in two or three months.

Death results most frequently from exhaustion; but pressure upon the trachea producing asphyxia is not uncommon, and death has occurred from starvation, the result of occlusion by pressure of the œsophagus. The complications already mentioned are the immediate causes of death in other cases.

Scrofulous enlargement of the glands presents the following points of difference: (1) Scrofula (tuberculosis) affects, as a rule, one group of glands, a local cause for whose enlargement is often present; (2) the glands tend to soften, with the formation of cheesy pus, and they may be somewhat painful; (3) it affects children much more frequently than is true of Hodgkin's disease; (4) the persons affected exhibit other manifestations of so-called scrofula, particularly in the eyes, nose, skin, and joints; (5) the blood changes, particularly the leucocytosis, do not reach the same intensity as in Hodgkin's disease; (6) the submaxillary glands are more

[1] British Med. Journ., April 30, 1892.
[2] Reynolds' System of Medicine, Philadelphia, 1880, vol. iii. 549.

frequently the seat of scrofulous adenitis, whereas Hodgkin's disease affects particularly the glands of the anterior and posterior cervical triangles.

Leucocythæmia is distinguished by the great enlargement of the spleen, the enlargement of the liver, and the characteristic blood changes.

Addison's Disease. Addison's disease is characterized by a gradual loss of strength without much loss of flesh; by gastric uneasiness and occasional vomiting; feeble circulation; and a bronze hue of the skin. The only fairly constant anatomical lesion is that of the supra-renal bodies.

The disease occurs most frequently during the active period of life, from twenty to forty, and nearly twice as often in males as in females. It is thought by some to be tuberculous in nature; some cases seem to have followed injuries.

The disease begins insidiously with gradual and progressive loss of strength. It becomes evident from the patient's languor, weariness on slight exertion, and inaptitude for mental effort that he is suffering with some exhausting disease. The appetite is impaired or lost, there is more or less discomfort at the epigastrium, and occasional vomiting.

Perhaps at this time a close inspection may show some discoloration of the skin, but usually this appears later. By degrees the gastric symptoms become more prominent, and vomiting may be so common as to shorten life materially. The most characteristic symptom is the extreme prostration without any obvious cause. Any exertion requires great effort, and may induce fainting. Finally, the patient is unable to leave the bed. Dull pains in the head, back, and abdomen are not uncommon; neuralgic pains in the limbs may be complained of; and Osler states that there is tenderness on pressure in the lumbar region in a considerable proportion of the cases.

The pulse is extremely small and feeble; in the later stages it may be absent at the wrist.

The discoloration of the skin is the most striking symptom of the disease when it is well marked. Sometimes the whole body becomes of a walnut-juice color, a bronzing which is deeper in exposed surfaces, as the face, neck, and hands, and wherever there is naturally a deposit of pigment, as the axilla and the genitals. At times only portions of the body are discolored, in which case the dark hue shades off gradually into the normal hue of the skin.

The pigmentation may extend to the mucous membranes of the mouth, eye, and vagina. Wilks[1] states that in all the cases which he has seen the scalp, finger-nails, soles of the feet, and palms of the hands escaped pigmentation.

Nevertheless discoloration of the skin is not an essential symptom of the disease; in some cases it is entirely absent. These cases, especially if associated with much vomiting, run a more acute course than the others, lasting only a few weeks. Such cases have been mistaken for typhus fever.

[1] Reynolds' System of Medicine, Phila., 1880, iii. 561.

The *diagnostic* symptoms are progressive asthenia, causeless nausea and vomiting, and bronzing of the skin and mucous membranes.

The *duration* of the disease is usually from six months to two years; but some have lasted from six weeks to ten years, and others, as already stated, prove fatal in a few weeks. Death results usually from asthenia, but it may also occur suddenly from syncope, or in coma and convulsions.

The differential diagnosis is from (1) jaundice; (2) pigmentation occurring in abdominal tumors; (3) pregnancy and chronic uterine disease; (4) melanotic cancer; (5) vagabond's disease; (6) leucoderma.

Exophthalmic Goitre. Exophthalmic goitre, Graves' or Basedow's disease, is a disease characterized by (1) great rapidity of the heart's action; (2) enlargement of the thyroid; (3) prominence of the eyes; (4) muscular tremor; (5) vomiting and diarrhœa, chiefly the latter, without cause.

It is far more frequent in women than in men. It may develop at any age, but is most common in early adult life. The particular cause is unknown, though it is probably located in the medulla. A neurotic heredity, exhausting disease, general debility, and anæmia are predisposing causes, while sudden fright or shock is the most common exciting cause.

Of the three classic symptoms, *rapidity of the heart's action*, with palpitation, *enlargement of the thyroid*, and *prominence of the eyes* (exophthalmos), the first is the essential symptom. It is also usually the earliest. Either enlargement of the thyroid or exophthalmos may be absent for months or years, and in some instances throughout the disease.

1. Graves' disease begins slowly. Attacks of palpitation may recur at intervals for a long time before their true nature is suspected. In these attacks the behavior of the heart is much like that which occurs under the influence of fright or great excitement. The frequency may not be over 100 or 120 in the early attacks, the rate being normal in the intervals. In the later and severe attaks, however, the pulse beats 160 or 180 or even 200. It is small and regular. The heart beats with increased force; the sounds are loud, sharp and clear, occasionally being heard several feet from the patient. In time the heart becomes hypertrophied and dilated, and there is often a loud basic systolic murmur.

The larger arteries and even sometimes the smaller ones show the vascular disturbance by increased pulsation, sometimes with thrill.

2. The thyroid is usually the next to become affected. It enlarges slowly from vascular dilatation, the swelling at first subsiding in the intervals between attacks, but subsequently persisting. The right lobe may be larger than the left. The enlargement is painless, soft, and compressible. It may pulsate with or without thrill, and over it can be heard hæmic murmurs.

3. Prominence of the eyes is the most conspicuous feature of well-marked cases. Like enlargement of the thyroid it varies in degree, and rarely is wholly absent. The protrusion allows the white sclerotic

to show above and below the cornea, giving the eyes an unnatural, startled, staring appearance. The protrusion may be so great that the eyelids cannot close; more commonly they close, but when the eyeball is simply directed downward the upper eyelids do not follow, but remain spasmodically elevated or lag behind the movement of the eyeball (Von Graefe's symptom). The eyeball may become inflamed and even slough from the undue exposure.

In addition to these characteristic symptoms the patient loses flesh and strength, has moderate pyrexia of irregular type, suffers from impaired appetite, diarrhœa, and despondency. The *diarrhœa* is of the nervous type—increased peristalsis without local catarrh. Menstruation is apt to be irregular or to cease. Tinnitus aurium, headache, and vertigo are not uncommon, and sometimes there is profuse sweating. *Muscular tremor*, occurring on voluntary movement, is frequently observed, and, with diarrhœa, is almost as common as the three primary symptoms.

Graves' disease, as a rule, runs a chronic course, lasting for years. A few cases that have run an acute course of a few weeks, some ending in recovery and some in death, have, however, been reported. Moreover, there may be recurring attacks with apparent recovery in the intervals. Recovery is thought to occur in about one-fourth of the cases. Gowers states that it is most frequent in the cases that develop rapidly and in which the cardiac symptoms preponderate over those in the neck and eyes, and that complete recovery is very rare when there is much enlargement of the thyroid and much prominence of the eyes.

Death results from gradual weakening of the heart and its direct and indirect effects. It may be hastened also by uncontrollable diarrhœa, acute mania, and epilepsy. The disease may also be complicated with hemorrhages, and these be the immediate cause of death.

Parasites in the Blood. The principal vegetable parasites are: (1) Spirilla of relapsing fever; (2) tubercle bacilli; (3) anthrax bacilli; (4) bacilli of glanders; (5) plasmodia of malaria; (6) typhoid bacilli.

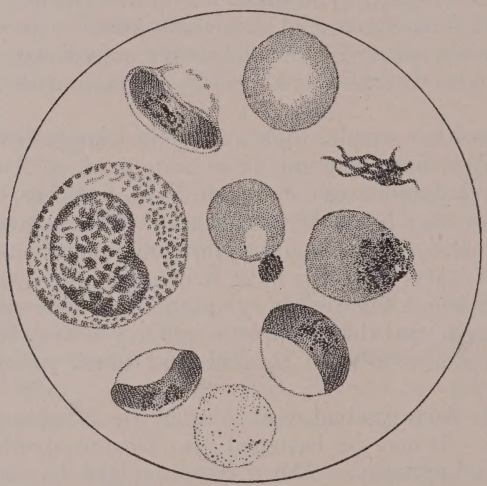
The animal parasites are: (1) Filaria sanguinis hominis; (2) distoma hæmatobium.

The Spirilla of Relapsing Fever. These are slender, thread-like organisms of spiral shape, seven or eight times the length of a red blood-cell, with a very lively forward movement in the direction of its long axis. Under a low power the blood may appear to be in motion, as the result of their movement. They have so far been found only in the height of the febrile attacks; but Von Jaksch states that so long as a relapse is to be feared the blood contains peculiar highly refracting bodies resembling diplococci, which are especially numerous before the attack; in some cases it has seemed to him that these diplococci at the very beginning of an attack develop into short, thick rods, from which the spirilla develop; they may, therefore, prove to be spores. Staining is unnecessary for the detection of spirilla, but cover-glass preparations of the blood can be stained with fuchsin or gentian-violet.

Tubercle Bacilli. Tubercle bacilli have been found in the blood in miliary tuberculosis. Cover-glass preparations of the blood are made and stained as in the case of sputum (which see).

Plasmodia of Malaria. The plasmodia of malaria were first pointed out by Laveran. They have been studied in Italy, especially by Marchiafava and Golgi, and in this country by Councilman, Osler, and Dock. Minute amœboid bodies are found first in the red corpuscles. These become pigmented with altered hæmoglobin, and grow until they fill nearly the whole of the cell, the pigment being arranged especially in a peripheral ring. Later, the amœboid bodies become spherical and

Fig. 130.



Malarial plasmodia. (Reproduced from colored plate.) Dry preparation. Stained with eosin-methyl-blue (one after the other). × 1600. To the right above, a normal rose-red stained red blood-cell, beneath it two such red-stained cells with bluish contained bodies, sprinkled with pigment, on one cell a colorless vacuole; next the same, bluish-stained parasite; farther away several of Laveran's crescents of clear violet or bluish color with reddish edge, which is continued in a fine line connecting both ends of the crescent, and which forms the remainder of the red blood-corpuscle. The pigment of dirty brownish-black color is constantly arranged in the centre of the crescent as a heap of fine granules. At the lower edge of the field is a colorless erythrocyst containing no hæmoglobin, and only containing a few scattered pigment granules. To the left a large mononuclear white blood-cell with large nucleus of bluish color. (H. Rieder.)

transparent, the pigment collecting in the centre. Sporulation now occurs and a fresh crop of small, rounded parasites appears, to begin the same cycle over again in fresh corpuscles. Golgi maintains that in tertian malarial fever the period between invasion of the corpuscles and the sporulation is two days; in quartan, three days, the difference in cycle being due to a difference in the parasites.

The onset of the fever corresponds in time to the division of the parasites.

The crescentic form described by Laveran is said to be more common in the irregular forms of malarial fever. Canalis[1] says that it only makes its appearance several days after the first access of fever. It is

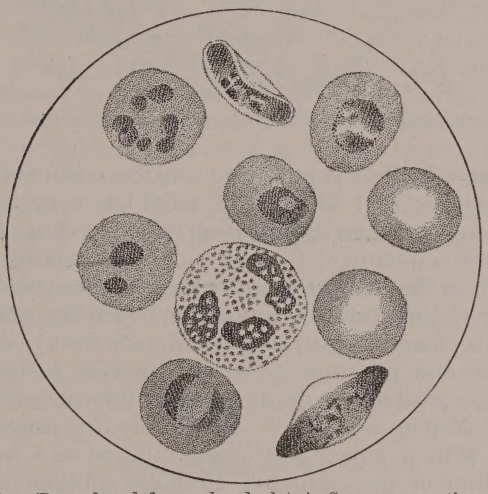
[1] Fortschritte der Medicin, 1890, viii. Nos. 8 and 9.

somewhat longer than a red blood-cell, and the pigment tends to collect in a focus about the middle of the parasite. Subsequently they become oval and divide into eight or more daughter cells.

Another form with flagella is occasionally found. Councilman says it is most common in blood drawn directly from the spleen.

The plasmodium of malaria may be *stained* as follows: Cover-glass preparations of the blood are dried in the air and fixed by immersion for twenty minutes or half an hour in a mixture of equal parts of alcohol and ether. They are then stained for ten to fifteen minutes in a solution of three ounces of strong aqueous solution of methyl-blue to which a few drops of absolute alcohol and then seven and a half grains of eosin

FIG. 131.



Malarial plasmodia. (Reproduced from colored plate.) Same preparation as preceding; same amplification. To the right two normal red blood-cells with central depression. In addition, several others with bluish contained bodies and pigment-sprinkled cells, which show the endogenous development of the plasmodia. Besides, two of Laveran's bodies, one exhibiting a delicate little basket. Near the centre a polynuclear white cell with bluish nuclei and red granulation. (H. RIEDER.)

dissolved in water are added. The cover-glasses are then washed in water, dried, and are then ready for mounting. The red blood-cells are stained rose, the nuclei of leucocytes a deep dark-blue, and any plasmodia a delicate sky-blue.[1] (See Malarial Fever.)

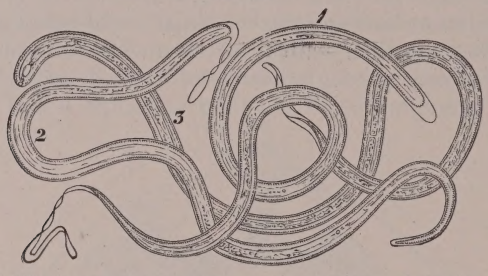
ANTHRAX BACILLI. Anthrax bacilli are found in small numbers in human beings with anthrax, especially in blood from the spleen. They are from 5 to 12 μ long and 1 μ broad, immovable rods, appearing as though divided into sections. They can be seen without staining, but the bacilli readily take the basic aniline dyes.

BACILLI OF GLANDERS. These are occasionally found in human blood. They consist of rods 2 to 3 μ long and 0.3 to 0.4 μ broad, frequently having spores on the ends. Löffler's staining method is recommended for their detection.

[1] Hochsinger: Wiener med. Presse, 1891, No. 17.

The Filaria Sanguinis Hominis. Filariæ are found in the blood and lymph of persons who live in the tropics, and in a few instances have been found in native Americans (John Guitéras). They have a blunt, rounded head with a tongue-like process and a long, pointed tail. They produce lymphatic swellings (particularly of the scrotum), chyluria, and hæmaturia.

Fig. 132.



Filaria sanguinis hominis—embryonic form. (Lewis.)

Patrick Manson[1] says the following are the commonest mistakes in the search for filariæ: (1) The use of too high a magnifying power; (2) employing too strong an illumination; (3) searching unmethodically and in too small a quantity of blood; and (4) looking for filariæ in blood drawn from the body at a time when the particular species sought for is normally absent from the circulation. He describes three forms: *filaria sanguinis hominis nocturna* (the ordinary form); *filaria sanguinis hominis diurna*, and *perstans*. The last appears to be the one associated with the production of the disease known on the west coast of Africa as "sleeping sickness." He prefers dry preparations of the blood, stained with a $\frac{1}{2}$ per cent. eosin solution or a weak solution of fuchsin (one drop of the saturated alcoholic solution to an ounce of water). If a thin film of blood, before it has fully dried, be held over acetic acid so as to imbibe the fumes, and be then stained in a $\frac{1}{2}$ per cent. solution of eosin, the blood is stained, but any filariæ remain pearly white.

[1] Trans. Seventh International Congress of Hygiene and Demography, vol. i. p. 93.

CHAPTER IX.

CONSTITUTIONAL DISEASES.

THE modern enthusiastic and voluminous labor in morbid anatomy and histology and in bacteriology has put into the background for the time being affections which, although they possess a distinct entity, are more vague and recondite. The occurrence of morbid processes behind which as a causal factor a constitutional state exists, as hemorrhages in hæmophilia or gastro-intestinal catarrh in rhachitis, must not be forgotten. The occurrence of abnormal phenomena, with or without a cognizable morbid process, should always call for the consideration of a possible general condition, or diathesis, as it was formerly termed, before the diagnosis is fully concluded. In a case of fever, for instance, we aim too often to determine the infection and its character, whereas an infective process may not be present, the fever being due to other, possibly constitutional, causes.

Advance in the science of medicine has transferred anæmia and chlorosis, formerly considered to be constitutional diseases, to the domain of blood diseases. Syphilis, tuberculosis, and probably cancer, are now known to be infectious diseases. The field has been narrowed; doubtless it will become extinct as our knowledge of constitutional affections becomes more precise.

Rheumatic Fever.

An acute, general, febrile, non-contagious disease, characterized by specific inflammation of the joints and their contiguous structures, hence called *acute articular rheumatism*. It is further characterized by a tendency of the inflammation to involve the larger joints successively, to skip from one joint to another, and to be associated with endo- and pericarditis.

The predisposing causes of rheumatic fever are *heredity*, which is operative in 25 or 30 per cent. of the cases; *age*—81 per cent. of first attacks occur between the eleventh and thirtieth years (Pye-Smith); *sex*, in childhood girls are more frequently affected than boys, but after that period sex appears to have no influence. Polyarticular inflammations, sometimes rheumatic in nature, are met with during convalescence from scarlatina and dysentery. They also occur in association with the puerperal state and gonorrhœa, in which they are probably pyæmic. The nature of the polyarthritis which occurs in connection with dengue and hæmophilia is obscure.

Damp, changeable weather appears to be more potent as an exciting cause than very cold weather. It is especially effective when the system is depressed from any cause. The disease occurs, however, at all seasons and in all climates.

Symptoms. The *onset* of the disease is not characterized by constant symptoms. Sometimes the fever and joint inflammation are preceded a day or two by debility, wandering pains in the joints or muscles, and loss of appetite. In other cases there is a chill or repeated attacks of chilliness, followed in a day or two by fever and inflammation of the joints. In rare cases the onset may be followed not by inflammation of the joints, but by that of the serous membranes, particularly those of the heart and its sac.

The *temperature* may rise a day or two before there are any joint symptoms, or fever and arthritis may begin almost simultaneously. The temperature rises rapidly to 102°, 103°, or 104 F., and one or more of the larger joints, generally the knee and ankle, become painful, tender, swollen, and hot. There may be great pain on motion before there is evident swelling or much local tenderness. The pain varies from discomfort to the most excruciating suffering. It is always aggravated by motion or pressure and is at times so exquisite that the slightest touch, the weight of bed-clothing or the jar of the bed from a heavy step in the room makes the patient cry out. It may extend beyond the joint to neighboring tendons and nerves. The swelling likewise varies greatly; sometimes there is only slight puffiness with increased distinctness of the cutaneous veins, increased heat in the part, but no general redness; in other cases there is considerable swelling about the joint so that the bony prominences are obliterated, the surface being tense, red, and very hot to the touch. There is often also effusion into the joint. Swelling is most marked in the wrist and ankle, and less so in the shoulders, hips, elbows, and knees.

Multiplicity of joints affected. A characteristic peculiarity of rheumatism is its tendency to involve one joint after another. One or several joints may be affected at first; it is very common for the right ankle to be affected, and then in a short time the opposite ankle, followed by the left knee and right knee, and so on with other joints. The inflammation usually lasts in each joint from two to four days. The process may subside in one articulation and begin in another with startling rapidity.

At one visit the right ankle may be swollen, hot, and unbearably painful, and on the next day the patient be found comfortable as to the first joint but suffering acute pain in the right knee or left ankle.

The *pulse* in the early stage of rheumatism is moderately accelerated (90 to 110); it is regular, of good volume, often bounding, and sometimes hard. The *urine* is scanty, high colored, abnormally acid, and deposits on cooling a copious precipitate of urates, resembling red sand in appearance. The *skin* does not feel so hot as one would expect from the temperature. It is continually covered with a copious, acid, and somewhat pungent perspiration. *Nervous symptoms* are not marked. There may, however, be slight nocturnal delirium. Sleeplessness from pain is very common.

The *temperature* in rheumatic fever is not usually very high; it is much oftener under 103° than over it. In rare cases, however, especially when the fever is complicated with pericarditis, pneumonia, or some disturbance of the heat-regulating apparatus, the temperature may attain the extraordinary range of 106–112° F. Such high tempera-

tures may occur suddenly or gradually, and are sometimes attended with marked brain symptoms (so-called cerebral rheumatism).

Endocarditis and *pericarditis* may occur at any period of rheumatic fever; they may even precede any joint-inflammation. They are most common, however, in the first two weeks of the disease. The younger the patient and the more severe the attack the greater the liability to heart complications. They occur in about one-fourth of all cases. Endocarditis is most common; often it is the only lesion, but sometimes it is associated with pericarditis, and more rarely with myocarditis. These complications usually give rise to no symptoms at first. Hence the heart should be examined daily. The occurrence of a sense of constriction in the præcordia or pit of the stomach, an anxious expression of the face with pallor, a change in the frequency but especially in the rhythm of the pulse, and of cough or dyspnœa, should attract attention to the heart. The physical signs of the respective lesions have been described fully under Diseases of the Heart.

The establishment of convalescence from rheumatic fever is marked by cleaning of the tongue, which also becomes less red, and the secretion of a large volume of urine, which still remains of high specific gravity. The fever subsides gradually, the joints cease to be red, swollen, and tender, the acid sweats lessen, and the appetite improves. In proportion to the duration of the case and its severity the patient is left with debility and marked anæmia, both red cells and hæmoglobin being diminished. In anæmic cases a hæmic murmur may be heard over the base of the heart. In some cases acute dilatation has been observed, and a tricuspid murmur.

Complications and Sequelæ. Apart from heart complications which have been mentioned already, *pleuritis*, *pneumonia*, and *bronchitis* occur in from 10 to 15 per cent. of the cases. They are frequently bilateral, and are very much more common in rheumatic fever with pericarditis or endocarditis than in simple rheumatic fever. Moreover, the pulmonary complications are frequently latent, and would be overlooked but for daily physical examination of the chest. Again, they may develop with great suddenness, and what appears to be a full-blown pneumonia may, on the other hand, subside suddenly as a fresh joint is affected. They behave more like sudden active congestions than true pneumonias. Rheumatic pleurisies are characteristized by the rapidity with which effusion takes place, the persistence of pain in the side during effusion, the tendency to involve both sides in succession, the readiness with which the effusion is absorbed, and their acute course.

Nervous System. The most common complications on the part of the nervous system are delirium, which is generally associated with insomnia and hyperpyrexia, but the latter is not constant. These brain symptoms generally appear in the second week of illness, and about the time of convalescence, or while the joints are still inflamed. The delirium may be low and muttering, accompanied by ataxic symptoms or even with tremors and spasms of muscles; or it may be furious. In favorable cases a deep sleep ushers in recovery, or in unfavorable cases the delirium persists with adynamia, the patient dying in collapse or coma, preceded or not by convulsions.

Chorea sometimes occurs as a complication, but it is more common in mild cases in children as a sequel. Cerebral meningitis occurs rarely, especially when there is ulcerative endocarditis. Cerebral embolism is another rare complication.

Various spinal symptoms occur in some cases, at times with and at times without demonstrable lesion of the cord or its membranes. Tetanus, myelitis, and spinal meningitis may all be simulated. Perhaps these symptoms are due to high temperature; but very high temperatures are met with without the occurrence of any cerebral or spinal symptoms.

Nephritis is rare, but sometimes hemorrhage into the kidney occurs with its usual symptoms. Peritonitis is extremely rare.

Various erythematous skin eruptions are seen from time to time, and occasionally purpura. Subcutaneous nodosities have been described by several writers. They are attached to the tendons, fascia, and periosteum, and are most frequent on the back of the elbow, the ankles, and patella. They are painless and may occur in any form of rheumatism.

The *duration* of the disease varies from a week to six or eight weeks, depending upon the severity of the attack, the presence or absence of complications, and the treatment. When the joint symptoms are pronounced and the fever continuous, the course is likely to be shorter. When the symptoms are milder, recovery may be retarded by repeated relapses.

Diagnosis. Rheumatic fever is distinguished from *gout* by the profuse acid and acrid sweating, the tendency to involve a number of joints and particularly the larger ones, by the greater intensity of constitutional symptoms, by the absence of uric acid from the blood, and the great liability to heart complications.

It is distinguished from *pyæmia* by the wandering character of the inflammation; the acid sweats; the absence of any antecedent condition liable to develop purulent foci—such as injuries, abscesses, or specific eruptive fever; the absence of chills, and the fact that in rheumatic fever the sweats are constant, whereas in pyæmia they follow a fall in temperature. Cutaneous abscesses do not occur in rheumatism, and upon its subsidence the joint's usefulness is not impaired.

Acute synovitis resembles rheumatic fever in there being pain, tenderness, and swelling of a joint. Usually, however, but one joint is involved, and there is a history of exposure to cold or of injury. The effusion is limited to the joint largely, is frequently abundant, and fluctuation can easily be detected. The constitutional symptoms are much less marked than in rheumatism.

Milk-leg or phlegmasia alba dolens differs from rheumatism in its occurring generally in women after confinement, or as a complication or sequel of fever, as typhoid fever. Usually only one leg is affected, or part of the leg, especially the calf. This becomes tense, tender, uniformly swollen, and the seat of great pain. The leg is moved with much difficulty. The femoral vein may be found to be knotted and tender. There is almost always evidence of antecedent disease.

Acute periostitis when close to a joint simulates rheumatism. But

the tenderness and heat are not in the joint itself, but are superficial and associated with less swelling. There may be detected by palpation some effusion under the periosteum. When this is purulent or there is also ostitis there may be chills and pyæmic symptoms.

The articular symptoms of *glanders* are to be distinguished by the occupation of the patient, the mode of onset, the associated symptoms, especially one or more pustules, and the fact that the painful joints are not so apt to be swollen and red as in rheumatic fever.

In *syphilis* there frequently occur joint pains, whose character is made out by the fact that the joints are not inflamed, the pain is much worse, or only occurs at night, and by the history of the patient and the therapeutic test.

In diseases of the brain and spinal cord joint inflammations occur of *trophic* origin. They are distinguished by the coexistence of some lesion of brain or cord, with hemiplegia or other palsy, and of other trophic changes, such as bedsores, atrophied muscles, loss or excessive growth of hair, shiny skin, and defective growth of the nails.

Subacute Articular Rheumatism.

In some instances the joint inflammation is less severe and is accompanied by only slight fever. One or more joints may be affected. It differs from the ordinary form in being milder in degree and more persistent, lasting sometimes for months. It is generally subacute from the beginning, but may be the type present in those who have had several attacks of rheumatic fever and have been left in a very sensitive condition. Rheumatic fever is usually subacute in children, and often only one joint is involved. Cardiac complications are more frequent than in adults, and chorea may occur as a sequel. Erythema nodosum and subcutaneous nodosities are more common in children.

Chronic Articular Rheumatism. In this form the patient has pain and stiffness in one or more joints, or in the contiguous tissues. The joints most frequently affected are the shoulder and knee. The pain is more or less constant, but worse in damp weather or on the approach of a storm, worse also frequently at night. Conversely, it is better in warm, dry weather. There is not much, if any, tenderness, and rarely any swelling or elevation of temperature. The joints very frequently crack and grate on motion. In the intervals of attacks there is no impairment of the usefulness of the joints. In very chronic cases there may be some atrophy of muscles and permanent stiffness, even fibrous ankylosis.

In some cases there are repeated attacks of subacute articular rheumatism accompanied with the usual symptoms and joint effusions.

The *duration* of the disease is indefinite, but it usually lasts for months or years; the patient becomes much debilitated from pain and stiffness. There is little risk to life, and cardiac complications are uncommon. It is distinguished from *chronic gout* by the fact that there is no special tendency to involve the great toe, by the absence of the deformities resulting from gout, and of deposits of urate of soda in the ears, fingers, and around the joints.

Muscular Rheumatism.

In this variety of rheumatism there is pain in the affected muscles, which often comes on suddenly in the night, or is first noticed when the patient attempts to rise in the morning. The pain when the patient is at rest may be inconsiderable, rarely amounting to more than a dull, aching, sore feeling; on attempting to move, to bend, or twist, or straighten himself, however, the patient catches himself suddenly on account of the agonizing tearing or burning pain. When the muscles are relaxed the patient is fairly comfortable. Sudden movement is the most painful. The affected muscles are tender to touch and to sharp blows.

Muscular rheumatism may be acute or chronic. In the latter the symptoms are very like those of chronic articular rheumatism, except that the muscles and not the joints are affected. There is the same proneness to recur in unfavorable weather and cold, damp seasons.

The disease receives different names according to the muscles affected. The most common sub-varieties are: *lumbago*, in which the muscles of the small of the back are affected; *pleurodynia*, in which the intercostal muscles suffer; and *torticollis*, in which the sterno-mastoid and trapezius are painfully contracted.

In lumbago the patient holds himself rigidly and is unwilling to rotate the trunk upon the vertebræ. Often the most comfortable position is that in which he sits and bends slightly forward over another chair. Motion is painful, but pressure is not. Fever is absent. There is a history of repeated attacks, or of exposure, such as lying upon damp ground. It needs to be distinguished from disease of the spinal membranes, from disease of the vertebræ, aneurism, abdominal abscess, and diseases of the uterus and ovaries. The diagnosis of rheumatism is arrived at by exclusion.

In pleurodynia there is usually tenderness upon pressure as well as upon motion and deep inspiration. The pain is of the same sore, burning character, aggravated by coughing and sneezing. The patient seems to breathe as little as possible, and often bends over toward the affected side to lessen the motion. It is distinguished from pleurisy by the absence of fever, cough, and, above all, of friction sounds. In intercostal neuralgia there are painful points upon pressure, whereas in pleurodynia, firm pressure is grateful, though tapping is painful.

In torticollis the head is drawn to one side and fixed in that position. The sterno-mastoid especially is rigid and tender on pinching. In spinal affections the head is retracted, and there are antecedent symptoms, as headache and darting pains with fever.

Rheumatoid Arthritis.

Rheumatoid arthritis or rheumatic gout is an affection characterized by acute or chronic inflammation of the joints, of progressive character, and resulting in deformities. It is accompanied with very little fever, and occurs apart from any known systemic disease.

It may be acute or chronic. The *acute* form differs but little in its

manifestations from acute rheumatic fever. Several joints are enlarged, tender, and painful. Constitutional symptoms, such as fever, loss of appetite, frequent pulse, thirst, furred tongue, occur as in rheumatism. Profuse acid sweats, however, are absent, and so is the tendency to serous inflammations. Moreover, while the larger joints, as in rheumatism, may be affected, the smaller ones also, especially of the fingers and toes, are inflamed and often the seat of serous effusions. Furthermore, the inflammation persists in the affected joints and does not jump from one to another. Instead of disappearing in a few weeks, it drags on for a much longer time. The pain subsides but the swelling persists, and permanent deformity results in at least some of the joints. The muscles of the arms and legs waste and are affected with painful spasms.

The disease is most common in young women exhausted by repeated pregnancies or prolonged lactation, and is favored by poverty, privation, and cold.

The *chronic* form is much more common. It also attacks most frequently young women who are exhausted or subject to great fatigue. There is pain, numbness, or formication in a joint, as the knee. The joint becomes tender, painful, and may be slightly swollen. This subsides after a while, but sooner or later the same joint or another one becomes affected, the process is persistent, one joint after another is attacked, and gradually all the joints may become greatly distorted, enlarged, and the seat of contractions. There may be no impairment of general health, or, at most, only dyspeptic symptoms. The progress is interrupted by remissions from time to time. Pain may be severe and subject to nocturnal exacerbations. The shape of the joints is altered by the effusion into the joints and adjacent bursæ, by thickening of the tissues around the joints, growths of new bone on the joint extremity of the bones, absorption of the articular cartilages and growths of new cartilage in the synovial sheaths, relaxation of the ligaments, muscular contractures, and luxation of the joints. The joints crack and creak like rusty hinges, are sore and stiff, and the attached muscles are affected with painful cramps.

Great enlargement of the joints at times occurs from the causes already mentioned and from infiltration of the overlying tissues. The enlargement is rendered more conspicuous by the atrophy of adjacent muscles.

In addition to the articular symptoms, other phenomena attend the process. One of the more common is increased *frequency* of the *pulse*. Although the patient is afebrile, the average pulse-rate is 100 to 120, or even more. Moreover, the pulse is soft and compressible, in contradistinction to the pulse of gout or rheumatism. It is worth noting that a return to the normal frequency is a sign that the process of the disease is arrested, although the joint lesions remain.

The *skin* is characteristic. It is soft and often much freckled, while the complexion is fair. C. T. Griffiths has observed the pigmentary cutaneous changes, along with neural symptoms, prior to the joint manifestations, and describes two forms: a diffuse melasmic discoloration, and dark-brown spots resembling moles, but not raised. Moisture of the skin with clamminess is common. It is limited to the palms of the

hands, or may occur in the distribution of certain nerves. The sweats are not acid; they are usually local, but may be profuse. *Pain* independent of the joint lesion is due to neuritis, and may precede the joint trouble. It is not merely confined to the nerve-trunks, but the distribution in muscles, as the base of the thumb. *Numbness* and tingling are often present.

The progress of the disease is pretty steadily worse. In extreme cases not only are the limbs crippled, deformed, and helpless, but there is fixation of the cervical spine and of the articulations of the jaw, so that the patient cannot move the head or masticate food.

The following describes the characteristic deformity of the hand: The first phalanx of the fingers is either flexed upon the metacarpus or extended, and the terminal phalanx in like manner is either markedly flexed or extended upon the second, or these two phalanges are kept at a straight line, while the first phalanx is, as usual, decidedly flexed upon the metacarpus. The hand is pronated and the fingers turn toward the ulnar side (Palmer Howard, and Charcot). (See page 127.)

The foot is abducted and flattened and the great toe abducted across and above the other toes. Rarely it may be beneath the other toes. The metatarso-phalangeal joint is enlarged.

A variety of the disease is sometimes met with, chiefly in old persons (senile arthritis), in which the tendency is to involve one or two joints, particularly the hip, or hip and knee. It is of slow progress and is otherwise attended with the same deformities as the usual polyarticular form.

Rheumatoid arthritis is distinguished from *gout* by the absence of heredity and by its development under the exhausting influences of repeated pregnancies, lactation, poverty, and malnutrition. Rheumatoid arthritis is progressive, with occasional remissions; gout occurs in successive attacks, with intermissions. Uric acid is absent from the blood in the former and is present in gout. Rheumatoid arthritis in the vast majority of cases is subacute or chronic. The acute form is distinguished from acute gout by the duration of the paroxysm and the absence of intermissions; by there being less heat, swelling, and redness of the joints, and less infiltration of the soft parts; by the fact that large and small joints are involved, and that there is no special tendency to inflammation of the great toe.

From *chronic gout* rheumatoid arthritis is distinguished by the absence of hereditary predisposition, of repeated acute attacks, and of the causes of gouty paroxysms—indulgence in sugars, acids, malt liquors, etc. Moreover, rheumatoid arthritis most frequently begins in the hands, and is symmetrical and bilateral. Gout has a predilection for the great toe, and is unilateral. Again, gout attacks well-fed males most frequently after the age of thirty, while rheumatoid arthritis tends to attack women under the depressing influences already mentioned. It may, however, occur in both sexes, and even be associated with gout.

Rheumatic fever is distinguished from acute rheumatoid arthritis by its tendency to involve the larger joints, its erratic course, acid sweats, and heavy deposits of urates from the urine, its shorter course, its ten-

dency to heart complications, and its subsidence without impairment of the usefulness of the joints.

Chronic articular rheumatism is distinguished by the preceding history, the tendency to seasonal exacerbations, by its involving fewer joints, and not being so symmetrical in the joints affected. It does not produce as great deformity as is common in rheumatoid arthritis, nor is it so likely to affect the vertebræ and jaws. The existence of valvular heart disease or a history of antecedent chorea is in favor of rheumatism.

The joint affections of *locomotor ataxia* are distinguished by the associated symptoms of incoördination and absent knee-jerk, by their sudden onset without pain or fever, by the occurrence of large effusion into the joint with subsequent disorganization, fractures, and dislocations.

Gonorrhœal arthritis is distinguished by the history of gonorrhœa or the existence of a discharge from the urethra, by the tendency of the disease to attack the larger joints, particularly the knee or shoulder, and to become fixed in one, not wandering from one to another. The affected joint suffers effusion, and the synovial membranes and bursæ are inflamed. The process is very chronic, but indolent, and the heart does not become affected.

Gout.

A disease characterized by specific arthritis, associated with uric acid in the blood and the deposit of urate of soda in the joints, or manifesting itself as a diathesis in which occur inflammations of non-articular tissues and various disturbances of functions of organs, the blood also containing uric acid.

Gout is common in Europe, particularly in England, but in its articular form is rare in this country. There is an hereditary predisposition in from 50 to 60 per cent. of the cases. It results from overeating of rich foods and the drinking of malt liquors, associated with deficient exercise and excretion. Garrod has called attention to its association with lead-poisoning. Paroxysms are induced by indiscretions in eating or drinking, by nervous shock or great mental strain, by exposure to cold or injury, or by overwork and sexual excesses.

The characteristic phenomena of gout are preceded for a variable time by acid flatulent dyspepsia, colicky pains in the stomach and bowel, constipation alternating with diarrhœa, and scanty, heavily loaded urine. Accompanying these dyspeptic symptoms often are impairment of physical and mental vigor, irritability of temper, and hypochondriasis.

In other cases the premonitory symptoms are palpitation of the heart, or dyspnœa resembling asthma, or various nervous symptoms, as drowsiness, insomnia, or headache.

In *acute articular gout* the onset is often sudden, especially in the first attack. The patient may go to bed in apparent health but be waked up early in the morning with a feeling of discomfort or uneasiness, usually in the great toe. In some cases the pain is agonizing from the first. The patient finds he is unable to step upon the foot without

torturing pain. The ball of the great toe is hot, swollen, red, and exquisitely tender, resentful of the slightest touch or jar of the bed. The veins are swollen and the joint stiff. There are slight fever, perhaps chilliness, thirst, coated tongue, constipation, scanty, high-colored urine depositing urates on cooling; the skin is warmer than normal and there is slight perspiration. The pain usually abates during the day and exacerbates at night, being aggravated by motion and with painful muscular cramps. By the end of the first day or two the swelling increases and the pain lessens, owing to diminished tension of the part. Pain is still great, however, on motion, and without treatment may continue for a week or two; under treatment the paroxysm subsides in four or five days.

Both great toes may be attacked in the first seizure, more often alternately than simultaneously, and sometimes other joints than that of the toe are affected.

With the subsidence of an attack the urine contains a larger quantity of uric acid, and the patient feels in better health and spirits than for some time. A second attack may be postponed for several years, but usually after that the intervals between them steadily lessen, until an attack recurs every few weeks or months, and the patient may be scarcely ever free from it. Other joints than the toes, particularly those of the fingers, become involved in subsequent attacks.

Chronic gout results from repeated acute attacks. It is characterized by deformity of the affected joints, around which are deposited chalk-stones (tophi) of urate of soda. Similar deposits occur in the helix of the ear. The first appearance is that of a clear vesicle under the skin, which subsequently becomes chalky white and solid. The deposits of urate of soda occur not only in the cartilages of the joints, but in the ligaments and bursæ also, resulting in great impairment of motion and deformity. "In extreme cases an appearance is presented by the hand very closely resembling a bundle of French carrots with their heads forward, the nails appearing to take the place of the stalks" (Garrod).

Gouty abscesses consist of collections of liquid and solid urate of soda, which discharge, with or without the presence of pus, through the skin. A patient may have a number of them with but very little impairment of the general health. They may even act as a helpful vent to the system.

Retrocedent gout is the name applied to the development of some acute internal affection upon the sudden suppression by cold or otherwise of an arthritis. Mania at times develops in this way.

Gout attacks the *nervous system*, causing headache, delirium, and sometimes apoplexy, apoplectiform seizures, epilepsy, mania, various neuralgias, and spinal symptoms.

It also affects the *heart* and *bloodvessels*, causing valvulitis and chronic arteritis.

The symptoms on the part of the *digestive organs* have been mentioned already. They are often premonitory of an attack.

The *kidneys* may be affected, causing typical contracted kidney, or there may be chronic *cystitis* and *urethritis*.

The *skin* gives evidence of its presence particularly in the form of psoriasis and eczema.

Rhachitis.

Rhachitis is a constitutional affection characterized by changes in the bones which lead to alterations in their shape and outline. It is usually developed in childhood, and is most common in children in bad hygienic surroundings, who have lived upon a starchy diet and have taken cow's milk for too long a period of time. A child that has been nursed during pregnancy is liable to have the disease.

The appearance of the face and the changes in the bones have been previously described (see page 76).

In addition to changes in the bones a child presents other evidences of defective nutrition. There is marked pallor; the muscles are flabby; the child is feeble, and the weak muscles give rise to an inaction which resembles paralysis.

The disease usually progresses slowly, and is eminently chronic. A form is seen, however, in which the progress of the symptoms is more acute. With some gastro-intestinal disturbances there is mild fever, considerable weakness, and great restlessness. Sleep is disturbed, and pain is complained of if the child is of an age to make such complaint. *Soreness* of the body is observed on handling the child; and of its own accord, on account of the pain and soreness, the customary movements are withheld. The child lies on its back and shrinks from any attempts to disturb it. The pain is not only caused by handling of the muscles, but the bones are also sore and tender. Sometimes the most marked manifestations of the more acute forms are the gastro-intestinal symptoms. It may often happen that vomiting and diarrhœa have as an underlying basis this rhachitic disposition.

With the above symptoms, and also in chronic cases, *perspirations* about the head are common. There is usually more heat of the head than is natural, hence in sleep the child rolls the head. This rolling causes the hair on the back of the head to be worn off. This sign is most characteristic of rhachitis when observed along with changes in the skeleton.

In the acute and chronic forms *enlargement* of the *liver* and *spleen* are observed. The enlargement is not only actual, but also a false enlargement may be seen from distortion of the organs on account of changes in the vertebræ and ribs. The abdomen is prominent, usually on account of flatulency, although the enlarged organs contribute to the swelling.

Of common symptoms in the course of rhachitis *nervous phenomena* are often observed. *Tetany* limited to the upper extremities, and *laryngismus stridulus* are the most frequent. Either of these complications may occur before the disease is otherwise suspected.

Diagnosis. The possible presence of rhachitis must not be forgotten in chronic vomiting in childhood. The disease must not be confounded with scurvy, which in children is likely to be the case. This is especially so in the acute form. It must not be forgotten that the latter affection may set in in the course of rhachitis. In scurvy, the pain, tenderness, and weakness are limited to the lower extremities. The immobility of the extremities may go on to pseudo-paralysis. The tenderness, however,

is great; œdema is more pronounced, and local areas of periostitis are more common. In scurvy the gums are swollen and may be spongy, or may be the seat of ecchymoses. The most decisive diagnostic criterion is the therapeutic test, scurvy rapidly yielding to a proper regimen.

Scurvy.

Scorbutus, or scurvy, is a constitutional condition brought about by a long-continued use of a diet deficient in fresh vegetables. It is characterized by pallor, great physical weakness and mental sluggishness, dyspnœa, subcutaneous and submucous hemorrhages, a swollen, spongy condition of the gums, and a brawny induration, especially of the calves and hams.

The onset of the disease is gradual, and is marked by a peculiar dirty-yellow or greenish pallor of the face, associated soon with an apathetic expression of the face, physical weakness, and decided lack of customary energy. The appearance is so characteristic that patients are said readily to detect it in others, though unaware of it in themselves. Sleep and digestion are good, but rheumatoid pains may be complained of. Other prominent subjective symptoms are fatigue on slight exertion, dyspnœa, faintness, and despondency. In the course of a week or two petechiæ appear upon the lower extremities, especially around a hair as the centre (see page 77). Depending upon the severity of the case there are also bullæ, vibices, and ecchymoses. Brawny induration, due to deep effusion of blood, occurs especially in the calves and hams, producing considerable pain on flexure of the knees.

There is no fever apart from complications. The pulse is frequent, weak, and small, and the first sound of the heart, and the impulse, may be very faint.

The face is swollen and of a dirty, possibly greenish-yellow color, according to Bird, Buzzard, and others; in some cases the eye and its surroundings are the only parts exhibiting signs of scurvy at this time. "The integument around one or both orbits is puffed up into a bruise-colored swelling. The conjunctivæ covering the sclerotic is tumid and of a brilliant-red color throughout, and about the eighth of an inch in thickness or elevation above the cornea, leaving the cornea at the bottom of a circular trench or well."[1] The condition is not inflammatory. These cases often terminate fatally.

Almost always the gums swell, become spongy, and bleed upon the slightest irritation. Sometimes they swell so as almost to hide the teeth completely, and even to protrude from the lips. The breath has a heavy, sickening odor, and the teeth sometimes drop out of their sockets.

In addition to the cutaneous and gingival hemorrhages, hemorrhages occur from the nose and other mucous surfaces, and effusions take place into the lungs, intestines, pericardium, and pleura, associated with inflammatory products. There may be no physical signs on the part of the lungs to account for the dyspnœa, or some dulness may be detected and bronchial breathing or a few râles.

[1] Buzzard: Reynolds' System of Medicine, 1880, vol. i. p. 451.

A very peculiar symptom, and sometimes the earliest, is hemeralopia, nyctalopia, or night-blindness, in which the patient can see during the day, but not by moonlight, and apart from artificial light is totally blind at night.

The prognosis is much better when there are external phenomena, even when they are very severe, than when these are absent. When there are marked pulmonary symptoms with tendency to syncope, the prognosis is grave. In general it is good if the disease can be brought under the influence of fresh vegetables and lemon-juice before it has seriously damaged the health.

The course of the disease is slow. Death may take place suddenly, and sometimes early, from syncope, but usually it is due to exhaustion, or to some complication, as dysentery, pneumonia (with or without gangrene), or ulcerative endocarditis.

Diabetes Mellitus.

The occurrence of any of the following conditions should lead to an examination of the urine for sugar, and an estimation of the quantity of urine passed in twenty-four hours, apart from the routine examination which should be made in every case of chronic disease or of obscure acute disease. 1. Muscular weakness without cause. The weakness is progressive and rapidly advances to an extreme degree. 2. Emaciation. In young subjects this is rapid in cases of diabetes. In older patients it is not so striking, particularly if the gouty diathesis is present. 3. Thirst. This is a symptom which is of common occurrence in diabetes, and is most distressing. If the amount of fluids taken be compared with the amount of urine excreted, it will be found that the two bear a definite ratio. The thirst is greater immediately after meals, although the patient does not necessarily have indigestion. 4. Hunger. Excess of appetite, boulimia or polyphagia, also occurs in diabetes. The amount of food that is taken is sometimes enormous, and the ravenous manner in which it is partaken of is revolting. 5. Loss of sexual power.

The four symptoms just mentioned, with increased frequency in micturition, are the common symptoms of diabetes mellitus. They may develop gradually. In rare instances the onset is sudden. The occurrence of these symptoms should lead at once to an examination of the renal secretion.

Three special characteristics of the urine are observed. *A.* The *amount* is increased so that from six to ten pints to thirty or forty pints are passed in twenty-four hours. *B.* The *specific gravity* ranges from 1025 to 1045, and may even be higher. *C.* The presence of *sugar.* The sugar is detected by the ordinary tests (see Examination of Urine). In addition the urine is usually of pale color, of a sweetish odor and acid reaction.

In addition to thirst and increased appetite, some gastro-intestinal symptoms may be of diagnostic importance. Of these, first, the appearance of the *tongue* is characteristic. It is dry, red, and glazed. The dryness is aggravated by a scanty flow of *saliva.* The *gums* are swollen

and spongy, and *stomatitis* is often present. There are no marked dyspeptic symptoms. *Constipation* is of common occurrence.

In diabetes other secretions are lessened in amount. *Perspirations* do not occur, except in inflammatory complications. The *skin* is harsh and dry. As the disease progresses the *heart's* action becomes weak and the *pulse* frequent and with lowered tension. The *temperature* of the body is usually below normal.

Diabetes may occur at any age, but is most frequent in adult life. In young adults the symptoms are more pronounced, and the duration shorter. In patients past middle life the disease may continue for a long period of years without marked interference with the health and nutrition.

While the symptoms just mentioned should lead to an examination of the urine, diabetes mellitus may not be suspected by any of the usual objective or subjective symptoms. It may happen that none of these symptoms are sufficiently marked, and that only by routine examination of the urine, or by the occurrence of affections known to be associated with sugar in the urine, is the disease discovered.

Of the complications which would lead to the suspicion of sugar in the urine the following are the most important:

1. *Cutaneous Complications.* Boils and carbuncles should always lead to an examination of the urine. Pruritus and chronic eczema may have diabetes in the background. Gangrene of the extremities, chiefly of the feet and legs, and gangrene in other situations, is of common occurrence in the course of diabetes.

2. *Lung Complications.* Tuberculosis, both of the chronic and the acute pneumonic type, is frequently associated with diabetes. Lobar pneumonia is liable to occur. In all cases of pneumonia the urine should be examined for sugar. Its presence would modify the prognosis of an otherwise moderate case. Gangrene is liable to ensue in the acute and chronic lung affections. Gangrene of the lung in the course of diabetes may be latent and recognized only by the odor and the character of the expectoration, or it may run an acute febrile course.

3. *Nervous Symptoms. Diabetic coma* is liable to develop in the course of the disease. In young subjects, particularly, the occurrence of coma should lead to a suspicion of diabetes. Such coma may occur before the disease has been recognized. The coma may follow an attack of fainting and prostration, with stupor, which deepens into complete unconsciousness. It may be preceded by nausea and vomiting or the lung complications previously mentioned. This form of coma is usually associated with extreme dyspnœa, and attended by agitation, pain in the head, and some delirium. The pulse becomes rapid and feeble, and coma develops gradually. For this form of coma the term *acetonæmia* is used. The breath is of peculiar sweetish odor, due to acetone, and this compound is detected in the urine. Coma may occur without any premonitory symptoms whatsoever, the patient reeling for a short time, and complaining of pain in the head as if intoxicated.

Peripheral neuritis should always lead to an examination of the urine. It may be localized to one group of nerves, or may be more or less general with symptoms like those of locomotor ataxia, as the

lightning pains, abolition of reflexes and loss of power in the extensor muscles. Diabetic patients are also subject to neuralgia, and to peripheral hyperæsthesia and paræsthesia, probably due to neuritis. The neuritis may be so extreme as to lead to paraplegia.

4. *Eye Symptoms.* A curious symptom of diabetes is the occurrence of *cataract.* This may develop at any age, and is often rapid in its course. Cataract or alterations of vision should always demand an examination of the urine. Diabetic *retinitis* is sometimes present. Atrophy of the optic nerves, or muscular insufficiencies, may take place, the latter causing the pronounced symptoms of eye-strain. Ringing in the ears, deafness, the occurrence of acute otitis, are phenomena which arise in the course of diabetes.

Diagnosis. Sugar in the urine occurs temporarily when there is an excess of saccharine diet, or when there is functional disorder of the liver. The sugar is small in amount, and the glycosuria is transient. The diagnosis of true diabetes is not difficult, although it may be overlooked unless the habit, previously insisted upon, of constant urinary examinations is fully developed.

Diabetes Insipidus.

This form of diabetes differs from the preceding in that the large amount of urine is normal, but of low specific gravity. The disease may come on suddenly after mental emotion, or develop gradually. The amount of urine may range from ten to forty pints. The urine is of low specific gravity—from 1001 to 1005. It is pale and watery. The solid constituents are not reduced. Urea is sometimes increased, but abnormal constituents are very rare. The passage of large amounts of urine induces thirst, but otherwise the symptoms do not tally with the symptoms of diabetes mellitus. The patients are usually well nourished.

The disease is usually secondary to some organic disease of the brain, or of the abdomen, as tuberculous peritonitis, abdominal tumors, or aneurisms. It usually occurs in males, and is often hereditary. It is most common in young people. Traumatism, meningitis, affections of the brain involving the sixth nerve, tumors of the brain or of the medulla, are causal factors. It may follow fright, a protracted spree, or perturbation of the nervous system from other causes.

The *diagnosis* is not difficult. It must be distinguished from the polyuria that is seen in chronic interstitial nephritis, and in amyloid disease. In hysteria, polyuria is common, although it is transitory. The presence of the stigmata and other hysterical manifestations lead to the diagnosis.

Hæmophilia.[1]

Hæmophilia is a constitutional affection characterized by bleeding, which is spontaneous or occurs upon slight injury. It is nearly always hereditary, but may arise *de novo.*

Males are very much more liable to it than females, the ratio being about 11 to 1. This curious disposition to bleeding may be transmitted

[1] See Hemorrhages, page 77.

for generations, and almost always to the males through the female members of the family—that is to say, the daughter of a bleeder is not usually affected, but she transmits the tendency to her sons, who become bleeders; her daughters are not bleeders, but they in turn transmit the disposition to their male offspring. It generally shows itself early in life, usually before the end of the second year, and almost invariably by puberty.

The affection usually first declares itself by the occurrence of a hemorrhage, either spontaneous or the result of slight injury, the bleeding being far more profuse than would be natural, and in some cases absolutely uncontrollable.

Legg[1] has divided hæmophilia into three degrees, according to the severity of the symptoms. The first is characterized by external and internal bleedings of every kind, and by joint affections; the second, by spontaneous hemorrhages from mucous membranes, but no traumatic bleeding or ecchymoses, and no joint affections; the third, by a tendency simply to ecchymoses. The first form is seen most frequently in men; the second most frequently in women; and the third in either sex.

The most frequent seat of hemorrhage is the nose, and the next the gastro-intestinal tract. The bleeding is from the capillaries; it may prove fatal in a few hours, or last for days and weeks with final recovery. Intense anæmia follows the prolonged hemorrhage, but the blood is replaced with remarkable rapidity. All operations, even the most trivial, are extremely dangerous in bleeders. Circumcision, extraction of teeth, and leeching are credited with the most deaths by Grandidier.

Joint symptoms are very common. The knees, elbows, ankles, and shoulders are the ones most frequently involved. The attack may be marked by pain, redness, swelling, inflammation, and fever; or fever may be absent; or pain alone be complained of. The attacks are liable to recur, especially in cold, damp weather, and may result in stiffened, deformed joints.

The *diagnosis* (see page 77) is easy when the history of an hereditary tendency to bleed can be obtained. Osler properly remarks that slight joint trouble and petechiæ are as much a manifestation of the disease as the more severe hemorrhages. In cases in which no such history can be got the diagnosis is made by noting a persistent liability to hemorrhage, without adequate cause, and associated with joint affections.

Osler gives the following excellent summary of the affections with which hæmophilia can be confounded:

1. The umbilical hemorrhages of infants, due to jaundice or to syphilis hæmorrhagica neonatorum, etc.

2. Purpura simplex, often seen in debilitated, rarely in healthy children, usually confined to the legs, and in some cases associated with rheumatic pains or swellings in the knees and ankles.

3. Peliosis rheumatica.

4. Purpura hæmorrhagica, morbus maculosus Werlhofii, a grave dis-

[1] Hæmophilia. London, 1892.
[2] Quoted by Osler, Pepper's System of Medicine, 1885, iii.932.

ease, characterized by extensive cutaneous ecchymoses, mucous hemorrhages, but not dependent on any local disease or, as far as is known, on any specific poison.

5. Infective purpura due to the action of some specific poison—smallpox, measles, scarlet fever, cerebro-spinal fever, etc. The hemorrhages may be cutaneous and trivial, or may be in the most aggravated form of interstitial and mucous bleedings, as seen, for example, in black smallpox.

6. Toxic purpura, as in snake-bites and many poisons, such as phosphorus.

7. Simple hemorrhagic diathesis, under which may be included those cases in which, without any hereditary disposition or previous hemorrhagic history, there is a tendency to uncontrollable hemorrhage from a slight wound.

8. Hæmatidrosis, bloody sweats, which occur usually in hysterical or epileptic females, and are in rare instances accompanied with mucous hemorrhages.

Purpura.

Secondary purpura occurs in connection with a variety of febrile and constitutional diseases: 1. Scurvy. 2. Hæmophilia. 3. Hodgkin's disease. 4. Splenic leucocythæmia. 5. Pernicious anæmia. 6. Chronic lesions of the kidney and liver. 7. Ulcerative endocarditis. 8. Malignant sarcomata.

Primary purpura occurs without any known cause. It has been divided for convenience into simple and hemorrhagic purpura, though the two probably differ only in intensity.

1. In simple purpura the hemorrhages are limited to the skin (see page 77). They consist of: 1. Bright-red spots, varying in size from a pinhead to a silver three-cent-piece. These spots are under the skin and are unaffected by pressure. They fade gradually from red to yellow and disappear. 2. Larger spots or streaks called vibices. 3. Ecchymoses.

The disease is said to be most common about the age of puberty. It may come on in the midst of apparent health, or it may follow an illness, as of typhoid fever.

Purpura occurs especially upon the legs, the standing position seeming to favor its occurrence. It comes out in successive crops. Sometimes large blebs filled with thin blood form under the skin, and gangrene at times occurs.

2. In the *hemorrhagic form*,[1] hemorrhages occur from the nose, stomach, bowels, vagina, and bronchi, or into the kidney or other viscus. Cutaneous and submucous hemorrhages also occur.

The onset of these cases is sudden, though there may be a day or two of depression, lassitude, headache, and nausea. The first symptom noticed is generally fever, which is apt to be moderate, then the eruption upon the skin is detected, and for a day or two the patient may seem to be only slightly ailing. Copious epistaxis may now occur, or a hæmatemesis or hæmaturia, or all of these and other hemorrhages may occur the same day. The temperature may be only moderately

[1] See "Grave Forms of Purpura Hæmorrhagica." Musser, Trans. Association of American Physicians, vol. vi.

raised, or it may reach 104° to 105° or even higher. The pulse at first is frequent (120 to 140), but of good volume and tension. Subsequently in unfavorable cases it becomes thready and very frequent. Respiration is not affected, and the mind is clear; the face is pale and anxious. Hemorrhage may also occur into the choroid and brain substance, with blindness and paralysis as sequels. It may also occur into the uvula or tonsil.

The *subjective symptoms* are pains in the loins, limbs, epigastrium, or chest. Often these pains announce a fresh hemorrhage, as into the kidney, or a fresh crop of purpuric spots. The degree of anæmia present depends upon the copiousness of the hemorrhage and the length of time the disease lasts. Sometimes the hemorrhages cause great exhaustion, with a tendency to collapse.

The urine, in the case of hemorrhage into the kidney, of course contains blood; sometimes casts are also found.

3. Another variety of purpura is known as *peliosis rheumatica*, the peculiar features of which are tender and swollen joints, œdema of the subcutaneous cellular tissue, and purpura associated with urticarial wheals and intense itching (purpura urticans). The subcutaneous hemorrhages consist of petechiæ, vibices, and ecchymoses. There may be such large hemorrhages into the penis, scrotum, and uvula as to result in gangrene and slow separation of the dead tissue by ulceration. Epistaxis may occur, but copious hemorrhages from the stomach, the bowel, or into the kidney or other organs are rare. Endocarditis and pericarditis occur as complications in some cases. The duration is apt to be tedious, convalescence being delayed by repeated outbreaks of purpura with multiple arthritic symptoms and œdema.

Diagnosis. It is distinguished from *scurvy* by the absence of antecedent debility and anæmia, of spongy gums, of brawny induration in the limbs, and by the fact that there is no tendency for the hemorrhages to occur around a hair follicle. In scurvy there is a history of deprivation of vegetable food, whereas purpura may occur in the midst of robust health. As a rule the cutaneous hemorrhages are larger in scurvy than in purpura.

It is distinguished from *acute infectious diseases*, particularly typhus, cerebro-spinal fever, and smallpox, by the absence of the severe constitutional symptoms which characterize the graver forms of these diseases—in which alone a purpuric eruption is likely to be severe enough to cause doubt. Hemorrhages from mucous surfaces are rare in the latter.

Hæmophilia is distinguished by the history the patient gives of being a bleeder by heredity, and the fact that the bleeding has been started by some injury, wound, or operation.

It is distinguished from the hemorrhages of *leukæmia* by the absence of enlarged spleen and liver, and by the fact that there is no excess of leucocytes in the blood.

Malignant sarcoma causing hemorrhages is recognized by the previous history of anæmia and cachexia, and by the detection of primary or secondary growths.

It must not be confounded with *Raynaud's disease*, a vasomotor affection characterized by local syncope, local asphyxia, and gangrene.

CHAPTER X.

THE INFECTIOUS DISEASES.

THE specific infectious diseases are those that are produced by a living contagion or micro-organism. The organism is introduced into the body through the skin, if the latter is the seat of some lesion, as in syphilis, tuberculosis, and anthrax; through the air-passages, as in diphtheria, scarlet fever, and other specific fevers; or through the digestive tract, as in typhoid fever, dysentery, and cholera.

The virus, as the living cause is named, in many instances produces certain changes at the point of entrance—the *initial phenomena*. It is then conveyed by the lymphatics or bloodvessels to near-by organs in the related lymph or blood stream, or transmitted to the whole body. When the whole body is affected sometimes an eruption is produced (eruptive fever), or the blood is changed in quality (diphtheria), or many tissues are affected simultaneously, or the nervous system notably disturbed. The above are the *phenomena of general distribution* of the virus, or of *infectiveness*. The virus or poison thus distributed may be the living organism, as in tuberculosis or anthrax, or it may be a poison generated by the organism, a toxin or ptomaïne, as in diphtheria.

Phenomena of secondary local distribution are due to local changes in organs affected secondarily. The poison has a special affinity for certain organs, as in whooping-cough, parotitis, pneumonia, or leprosy.

In some instances the local phenomena are so marked as to give to the disease a corresponding distinctive feature. They are the granulomata.

Bearing in mind the above distinctions, specific infectious diseases are divided into six classes.

FIRST CLASS. *Acute Specific Fevers.* The initial phenomena are slight. The phenomena of infectiveness are marked; an eruption is one of the most characteristic. The secondary local phenomena are variable. The following are included in this class: Typhoid fever, typhus fever, variola, varicella, scarlet fever, measles, relapsing fever, plague, and cholera.

SECOND CLASS. *Specific Inflammation.* Initial phenomena indefinite. General phenomena (infectiveness) variable, but no eruption. Specific affinity of poison for one particular structure. Whooping-cough, mumps, diphtheria, dysentery, erysipelas, tetanus, hydrophobia, pneumonia belong to this class.

THIRD CLASS. *Contagious Suppurations.* Initial phenomena marked (suppuration); generalization not marked unless the virus enters the blood; secondary local phenomena decisive. Gonorrhœa is one type, pyæmia a second, in which the blood is infected.

FOURTH CLASS. *Infective Granulomata.* Distinct initial phenomena.

Phenomena of generalization not marked, or like specific fevers. Secondary local phenomena prominent. Examples: Tuberculosis, syphilis, leprosy, and glanders.

FIFTH CLASS. *Miasmatic Diseases.* No initial phenomena.

SIXTH CLASS. *Vegetable Parasitic Diseases.*

The infectious diseases, as pneumonia and dysentery, not included in this section, are considered under local diseases as a matter of convenience.

Typhoid Fever.

An acute, specific, infectious and mildly contagious fever, characterized by a gradual onset, a continued fever, an eruption of rose-colored spots, marked nervous and abdominal symptoms, and an average duration of three or four weeks.

It occurs sporadically and epidemically, and in large cities is apt to be endemic. Its special habitat is in temperate climates, but it may occur anywhere. It is relatively rare in the southern and southwestern portions of the United States. It is more frequent in the latter part of the summer and in the autumn and winter, and following hot and dry summers. Young adults are especially prone to it, but cases have occurred at all ages. Change of residence from the country to the city predisposes to it. Those living in cities often acquire immunity, but they may lose it upon moving elsewhere. The state of previous health does not seem to have any influence.

In by far the larger number of epidemics the poison has been conveyed in the water, in a few instances in the milk previously contaminated by water. In sporadic and endemic cases the poison may be obtained from defective house drainage and from damp, unwholesome cellars. The specific cause of the disease is believed to be a bacillus described by Eberth and others.

The period of *incubation* in typhoid fever varies from four or five days to three weeks; more commonly it is from one to two weeks. During this time the patient usually is languid, becomes tired easily upon exertion, has severe headache, and sleeps poorly. There is often, even thus early, a dull and listless expression of the face. Toward the close of this period, and in severe cases, there may be colicky pain in the abdomen, a tendency to looseness of the bowels, cough, epistaxis, mental sluggishness, and chilliness. Dr. Pepper says he has been led repeatedly to anticipate the approach of typhoid fever by the unusual dulness of hearing and by the persistent occipital headache coming on after a few days of general malaise.

While the disease may begin abruptly, a gradual onset is so much the rule that it becomes important in the diagnosis from other disease conditions.

Invasion is not sharply marked. There may be chilliness, but a decided chill is unusual except when pneumonia is part of the initial process. Muscular weakness, headache, and mental sluggishness are more pronounced, and the physician is consulted because these symptoms persist, or because fever is discovered. The beginning of fever is the most constant indication of the onset of the disease, and two

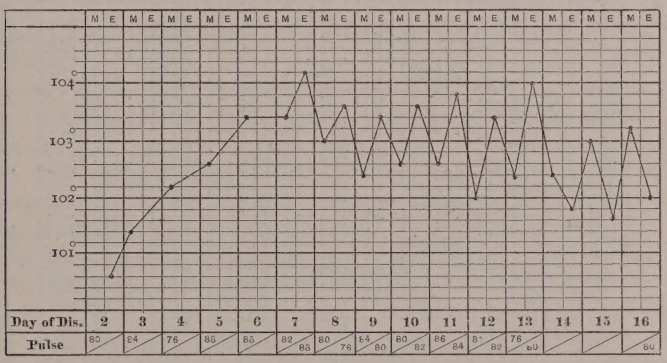
very important early symptoms are cough and enlargement of the spleen.

The most prominent and constant subjective symptom during the first week is headache. Other very common symptoms are tenderness, rarely pain, in the iliac region, more or less prostration, and impaired or lost appetite.

The *objective* symptoms are therefore the most important. The face is pale rather than flushed, and has a dull, listless, apathetic expression upon it. The tongue is heavily coated with a white fur which becomes yellow. The abdomen is somewhat distended and tympanitic on percussion. There is usually tenderness in the right iliac region, and gurgling upon palpation is pretty constant. Constipation may be present at first, and sometimes persists throughout the disease. A tendency to diarrhœa is, however, characteristic of the disease. Even if constipation exist at first, a laxative is apt to produce an excessive effect. The number of passages varies from two or three to a dozen or more in twenty-four hours. They are light yellow in color ("pea-soup"), thin, watery, and offensive. The movements are not usually accompanied with pain, but in severe cases may occur involuntarily.

Enlargement of the spleen is a very constant symptom. It may be detected at the onset, increases up to the height of the fever, subsides during convalescence, but recurs during a relapse. It covers a percussion area in the left hypochondrium of four to eight finger-breadths.

FIG. 133.



Mild typhoid fever. Gradual ascent.

The *temperature* curve when not modified by treatment shows a gradual ascent during the first four or five days of the disease, with a morning remission. The temperature rises a degree or two in the evening and falls half a degree or a degree in the morning. This "step-ladder" ascent is very characteristic. By the end of the week a temperature of 103°, 104°, or 105° has been reached, and it remains continuously high, with slight morning remissions, during the second, and less frequently during the third week. In the third or fourth week the morning fall of temperature gradually becomes greater, and by the end of the week sinks below normal in the morning.

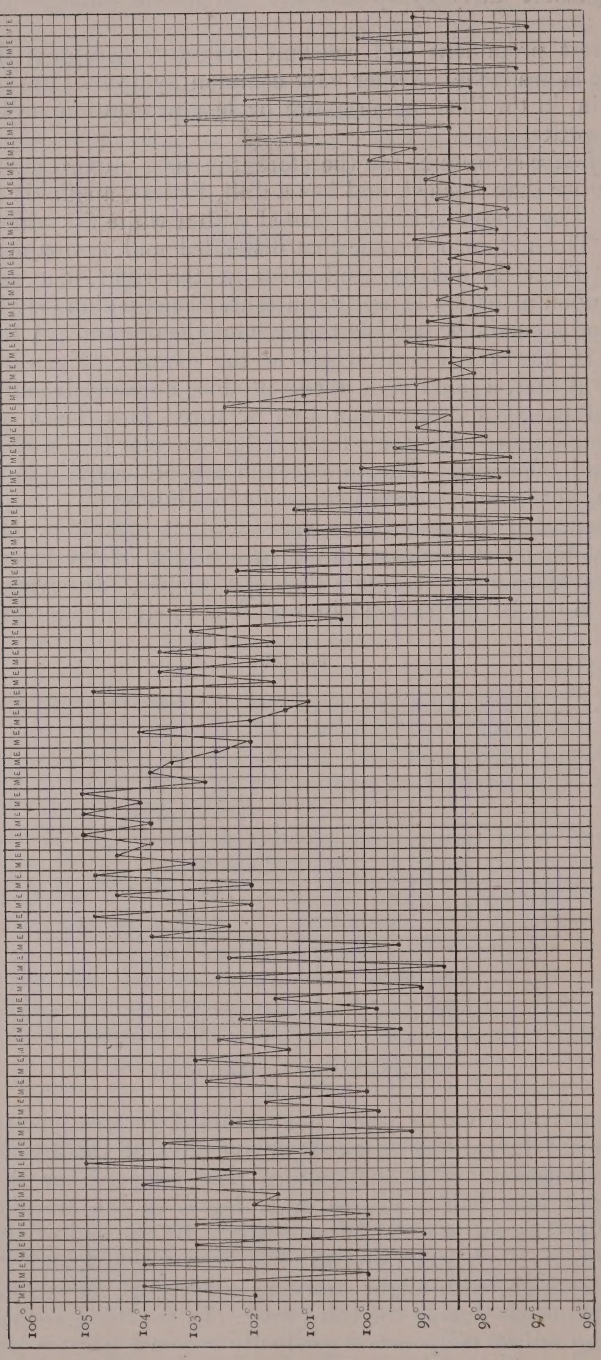


FIG. 134.

Protracted typhoid fever: not modified by antipyretics. Aberrant temperature during first twenty days. Duration, 63 days. Recrudescence on the fifty-fifth day.

The temperature in mild cases may never rise above 103° at any time, and most of the time varies between 100° and 102°. Or it may be 104° from the start; more frequently during the second and third week there are marked oscillations of the temperature—a sudden fall from 104° to 101°, or a rise from 103° to 105° or 106°. Hyperpyrexia is a temperature above 105°.

The *pulse* is full, and in favorable cases slower than the pyrexia would lead one to suppose. It is more frequently under 110 than over 120. In the second week it is markedly dicrotic.

The *heart sounds* are unchanged apart from complications, but in the second and third weeks the first sounds often are feeble, indicating heart weakness. A pulse of 120 or more is a graver sign in typhoid fever than in other diseases. Therefore when it becomes very frequent and feeble, the extremities cool and the lips bluish, the outlook is gloomy.

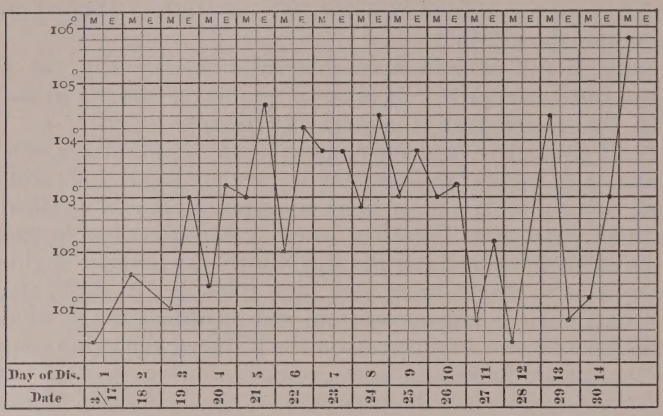
The *urine* is at first scanty and high-colored. A slight degree of febrile albuminuria is not uncommon, and in rare cases the whole force of the poison seems to be spent upon the kidneys, the urine containing, besides the usual blood and casts, biliary coloring matter. In conditions bordering on coma the patient may have retention of urine, or, on the other hand, he may pass it involuntarily. The diazo reaction of Ehrlich is obtained by mixing forty parts of a one-half per cent. solution of sodium nitrite with one part of a one-half per cent. solution of hydrochloric acid saturated with sulphanilic acid. Equal volumes of the mixture and of urine are shaken up in a test-tube and covered with ammonia. At the junction of the two a pink or ruby ring develops. This reaction is helpful in diagnosis, but may occur in acute phthisis, tubercular meningitis, and other diseases. According to Pepper it is rarely absent in measles.

The *respiration* in uncomplicated cases increases in frequency with the rise in temperature. It usually ranges between 24 and 36. The slight bronchitis present in the beginning in most cases causes no trouble; sometimes it lasts throughout and contributes to the tendency to hypostatic congestion always present. The physical signs are those described elsewhere in these conditions.

The *nervous symptoms* are often very prominent. In mild cases they consist of hebetude and nocturnal delirium, or they may be absent altogether. Usually, however, by the beginning of the second week there is some mental confusion with nocturnal delirium. In more severe cases and later in the disease the delirium is of a low, muttering character, with hallucinations of sight and sound more or less continuous. The patient can be roused by a question, and makes an intelligent answer, but speedily lapses into semi-consciousness. Picking at the bedclothes or efforts to catch imaginary objects are very common. Sometimes the delirium is wild and noisy, and the constant presence of some one is needed to keep the patient from getting out of bed. Patients have jumped out of windows, or run long distances before being captured. Rarely the delirium has been so active as to simulate acute mania. Stupor may alternate with delirium. Rarely the patient lies with wide-open eyes, apparently staring fixedly at some object, but really unconscious (coma vigil).

In ataxic cases the patient has marked twitching of the tendons and jactitation. He is wakeful and restless, wearing himself out. The

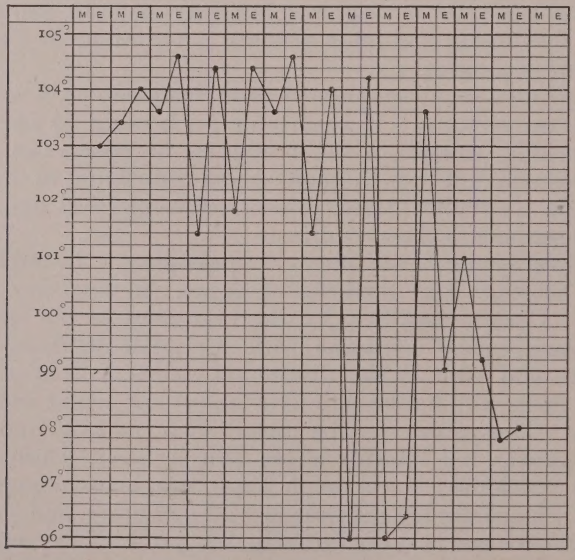
FIG. 135.



Grave typhoid fever. Death. M., æt. 22. Ataxic symptoms.

hands and lips tremble, and he keeps muttering to himself all the time.

FIG. 136.



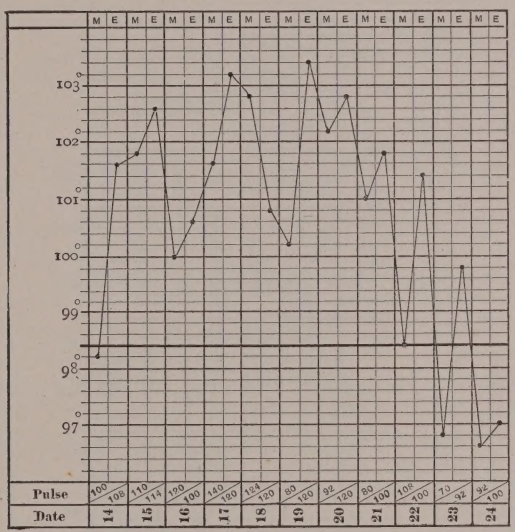
Typhoid fever in a child æt. 12. Chart from twelfth to twenty-third day. (Frequent mode of termination in children.)

Convulsions are rare, but may occur in children. Sometimes there is considerable hyperæsthesia and tenderness along the spine.

The extent of the nervous symptoms depends upon the habit of the patient as well as upon the height of the temperature and gravity of the disease. They may be pronounced in children and neurotic individuals with moderate fever.

On the seventh or eighth day the *eruption* appears. It consists of small, very slightly elevated, rose-colored papules, which disappear upon pressure and come out in successive crops, each papule lasting three or four days. The spots are most common over the abdomen and back, but are occasionally found elsewhere. They are usually few in number, a half-dozen or dozen, but sometimes the eruption is very copious. This is more apt to be in severe cases. Sometimes it is wholly absent.

FIG. 137.



Course of temperature in a relapse beginning on twenty-sixth day. First attack mild.

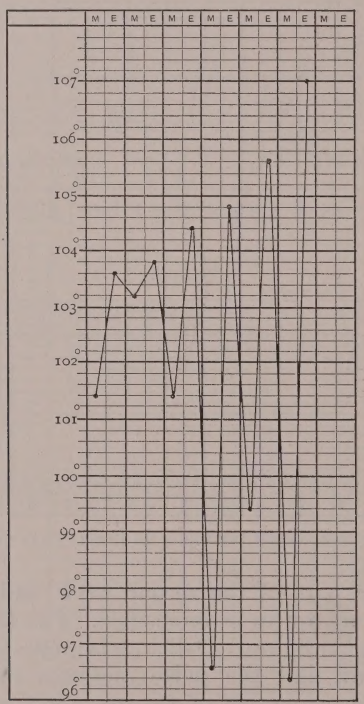
During the latter part of the second week and through the third week the symptoms are apt to be intensified. The temperature keeps up or even reaches a higher figure. Delirium is more decided and constant. The heart grows weak and the pulse increases in frequency. Some degree of hypostatic congestion of the lungs is usual. Diarrhœa may be troublesome; intestinal hemorrhages, announced by sudden fall of temperature and symptoms of collapse, may occur. Tympanites may become so great as to interfere with respiration and circulation. This is the period when ulceration of Peyer's patches in the intestine is deepest, and perforation is imminent. There is rarely any desire for food, though it is taken and assimilated. Nausea and vomiting are rare. But the tongue is dry, brown, sometimes glazed and fissured, and sordes often collect on the teeth.

In cases ending in recovery the temperature begins to fall in the mornings; delirium grows less; sleep is more refreshing. Diarrhœa

ceases, and constipation may even require treatment. The pulse does not usually improve as rapidly as the other symptoms. There is sometimes very marked anæmia without leucocytosis (Osler). When the temperature sinks to normal or subnormal, convalescence has set in. This is very rapid as far as digestive symptoms are concerned, but the strength returns very slowly. It may be interrupted by a relapse, in which the original symptoms are reproduced, with high temperature but of shorter duration.

VARIETIES. The abortive form is so named because of the abbreviated course of the disease. The symptoms are sufficiently well marked to make the diagnosis clear, but the type is mild, and in a week or two convalescence is established.

FIG. 138.



Grave typhoid fever. Daily rigors. Death on nineteenth day. No complications.

In the ambulatory form, commonly called "walking typhoid," the patient, from ignorance of the gravity of his ailment or from apparent necessity, keeps at his work until weakness and incessant headache lead him to consult a physician in his office or at a dispensary. He may then be well into the second week of the disease. The majority of such cases prove fatal.

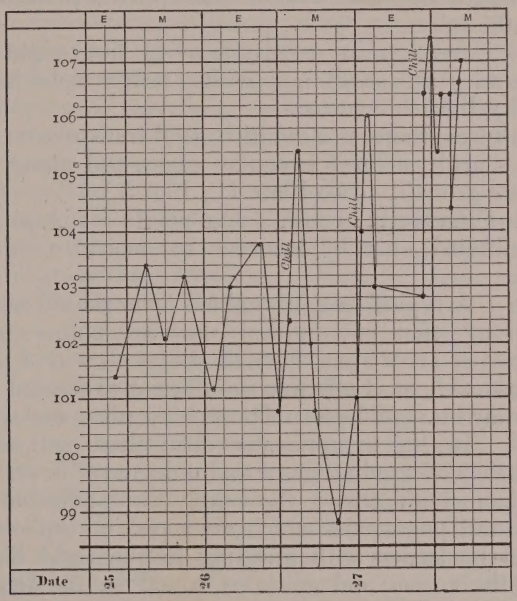
In the pulmonary form the onset may be so obscured by severe bronchitis or lobar pneumonia that the primary disease is not suspected

at first. Severe bronchitis seems to be more common in children. Chill and initial high temperature are common in these cases.

Grave forms are due to especial severity of some symptom or group of symptoms, such as hyperpyrexia; profound stupor, coma, or intense ataxia; inability to take or retain sufficient nourishment; profuse diarrhœa and intestinal hemorrhage; great adynamia with weak heart and a tendency to cyanosis. In other cases the gravity results from the existence of complications.

In the malignant form there has been a large dose of the poison or a very weak organism, or both, the result being an acute toxæmia; this is not so common as in scarlatina and typhus fever. Other relatively rare

FIG. 139.



Renal typhoid. Nephritis on the twenty-fifth day. Course of temperature during three days preceding death.

forms are the renal and afebrile. Typhoid fever may be accompanied by a number of complications, the most frequent and important being severe bronchitis, hypostatic congestion with œdema, and true lobar pneumonia; bedsores; parotitis; phlebitis, especially of the femoral vein; peritonitis from perforation of the bowel; meningitis, acute mania, or mental decay; jaundice; myocarditis; periostitis and osteitis. Sequelæ are not frequent. Sometimes, however, the foundation is laid for permanent ill health. There may be impairment of the senses, mental weakness, and even insanity. Paralyses, neuritis, hyperæsthesias, chorea, and epilepsy are occasional sequels.

BACTERIOLOGICAL DIAGNOSIS. *Eberth's Bacillus.* The bacillus is found in colonies in the spleen, liver, mesenteric glands, kidneys, and

intestines of cases of typhoid fever. It is also found in the fæces and rarely in the urine. It may be seen in the blood.

Morphology. A bacillus 1 to 3 μ long by 0.5 to 0.8 μ broad, with rounded ends. It is motile, facultative anaërobic, does not liquefy gelatin. It has flagella 3 to 5 times as long as the bacilli. It stains with the anilines, best with Löffler's blue. The flagella are stained by Löffler's special method. (See Plate I., Fig. 6, B.)

Biological Properties. It grows readily in acid media as well as in the neutral or alkaline media, best at a temperature of 38° C. (Death-point, 60° C.)

The organ from which a culture is to be made is washed carefully in a bichloride of mercury solution. Then three cuts are made with different sterilized knives, the third cut reaching the central part of the organ. A little of the tissue is then taken with a platinum needle and inserted into the tubes.

The colonies develop in twenty-four to forty-eight hours. On gelatin plates they are small and white, nearly spherical; irregular, granular, and yellowish brown.

In stab cultures there is a whitish semi-transparent layer on the surface with sharply defined irregular edges, and along the puncture a grayish-white growth. (See Plate II., Fig. 5.)

It develops abundantly in milk. On potato it forms an "invisible growth" manifested only by increase in moisture, which is quite characteristic.

DIAGNOSIS. A typical case of typhoid fever should not be mistaken for any other affection, but atypical cases are numerous. The most common sources of error are a hurried diagnosis and a willingness to accept a demonstrable local affection as sufficient to account for the condition. In this way the significance of bronchitis, pneumonia, and diarrhœa is overlooked. In the symptomatic form there will almost always be found a history of gradual onset and a degree of fever and prostration greater than should attend the purely local affection. Moreover, in bronchitis and pneumonia which are a part of typhoid fever there may be found tenderness with gurgling in the right iliac region, enlargement of the spleen, and epistaxis, to aid in the diagnosis; while in cases in which the diarrhœa leads to uncertainty, bronchitis, enlargement of the spleen, and epistaxis may coexist.

New Diagnostic Sign of Typhoid Fever. Dr. Simon Baruch writes as follows: "As soon as a patient shows a rectal temperature above 102.5° in the morning and 103° in the evening for three successive days, especially if this be accompanied by headache, dulness, or apathy, he is placed in a full bath at 90°, which is reduced to 80°, with constant friction over the body. In three hours, the temperature still being above 102.5°, he receives another bath 5° cooler. This is repeated until the temperature of the bath is 75°. If one or more of these baths fails to reduce the rectal temperature 2° in half an hour, the diagnosis of typhoid fever is almost certain, and the bath treatment is continued. The resistance of the rectal temperature to a bath of 75° for fifteen minutes, with friction, is an almost certain test of typhoid fever."[1] Dr.

[1] New York Medical Journal, September 2, 1893.

Baruch considers that the diagnosis of this disease should no longer be obscure, even in the first days of its course.

Appendicitis is more likely to be mistaken for typhoid fever than the contrary. There is usually a history of constipation, though the occurrence of several inadequate movements a day may conceal the fact that there is a fæcal accumulation. The onset is more abrupt and the local symptoms more pronounced than in typhoid. Pain and tenderness are prominent, and while they may be general over the abdomen at first, they are found to be more acute in the iliac region and loin. Here, in place of gurgling, we find some increase of resistance on palpation, and a relatively dull note—a wooden sort of tympany—or there may be a demonstrable tumor. The patient lies with the right leg drawn up, has moderate fever, and vomiting. In fact, the attack is often introduced by chilliness and vomiting. Headache is not a prominent symptom, while bronchitis and enlargement of the spleen are absent.

Acute right-sided *salpingitis* simulates typhoid fever. It is distinguished by the history of a preceding vaginitis, endometritis, or abortion, by the absence of diarrhœa, enlargement of the spleen, and the characteristic eruption. A digital examination through the vagina discovers the womb pressed to one side and fixed, and a tender mass blocking up the pelvis.

Simple continued fever is distinguished from typhoid fever of a mild type principally by the absence of bronchitis, enlargement of the spleen, epistaxis, and characteristic eruption. Constipation is more common than looseness of the bowels, and gurgling is absent.

Typhus fever is distinguished by its sudden onset, the besotted expression of the face, with reddened eyelids and small pupils, the absence of abdominal symptoms, and the occurrence on the fourth day of maculæ, which are subsequently converted into petechiæ. It is of shorter duration, and terminates very abruptly by crisis.

Relapsing fever differs from typhoid fever in its sudden onset with chill, pain in the epigastrium, but absence of abdominal symptoms and eruption; in the absence of marked nervous symptoms, in spite of the high fever; the short duration and termination by crisis, and characteristic relapse at the end of a week. The conclusive test is finding spirilla in the blood.

Acute tuberculosis of the lungs, at times, closely resembles typhoid fever. In both the onset is gradual, with cough and fever. In the former, however, the bronchial symptoms are more prominent, there are apt to be recurring chills and sweats, the temperature is remittent and irregular, emaciation is rapid, and constipation instead of diarrhœa is the rule.

In *peritoneal tuberculosis* there is persistent pain in the abdomen, which is general; the belly is swollen. If effusion occurs, the percussion note is dull. The temperature is irregular and may be below normal; nervous symptoms comparable to those of typhoid are wanting.

Meningitis before the stage of effusion exhibits exaggeration of the reflexes and marked hyperæsthesia. There may also be muscular rigidity. The patient is restless, easily annoyed, and "fussy" about things that would be unnoticed by a typhoid patient. Vomiting is

often present, whereas it is rare in typhoid fever. The temperature does not maintain so high an average range as in typhoid and is subject to greater oscillations. The pulse varies greatly, and may be irregular.

In *septic meningitis* the headache and vomiting are more persistent, the bowels are confined, and the abdominal walls are retracted. There may be double optic neuritis. In *tubercular meningitis* the knee-jerk and other reflexes are variable, irregularly absent or present. In typhoid fever they are always present. In the former, choroidal tubercles may be seen with the ophthalmoscope. In *tuberculosis* in all forms leucocytosis is present; in typhoid it is absent. Typhoid fever must not be confounded with *trichiniasis*; the peculiar muscular pain and œdema do not occur in the former. *Uræmia* may simulate typhoid fever when it becomes chronic, but the age, the characters of the urine, the cardio-vascular symptoms, are diagnostic, and with the absence of the specific typhoid symptoms render the diagnosis easy.

Typhus Fever.

An acute contagious and infectious fever, occasionally occurring sporadically and liable to be epidemic in the presence of destitution, filth, overcrowding, and bad ventilation. It is characterized by abrupt onset with chill or chilliness, a rapid rise of temperature, lassitude, headache, and pains in the back and limbs. On the fifth day a peculiar spotted eruption appears, which at first is macular and subsequently petechial. It is further characterized by adynamia or ataxia, low muttering delirium, a suffused, heavy, drunken expression of countenance, by the absence of local disease, and by a crisis which occurs on or about the fourteenth day.

Typhus fever is variously known as *ship-fever*, *jail-fever*, *camp-fever*, etc., names which sufficiently indicate its tendency to develop in the presence of filth, overcrowding, and privation. It is rare in this country, but is occasionally introduced at our seaports.

The period of *incubation* is usually about twelve days; it may be five or eight days, or even a shorter time, depending upon the virulence of the poison and the susceptibility of the patient. Malaise may precede by a day or two the onset of the disease.

Invasion is characterized by headache, faintness, vertigo, chilliness, or a distinct rigor, pains in the back and thighs, loss of appetite, nausea, constipation, and extreme weakness. The prostration is sometimes so great as to compel the patient at once to go to bed. The *temperature* rises rapidly to 104° or 105° at the end of the second or third day. The *pulse* is frequent, 100 or 140, and in grave cases shows a marked tendency to become small, soft, and feeble. The patient is restless and sleepless, and is annoyed by tinnitus. The *expression* of the flushed face is listless and dull.

About the fourth or fifth day the typhus *eruption* begins to appear. It consists at first of dull-red spots of irregular size and shape. They are most numerous on the covered parts. Moore[1] says they are

[1] "Eruptive and Continued Fevers," by J. W. Moore, Dublin, 1892.

detected first near the axillæ and on the wrists, then on the sides of the abdomen, afterward on the chest, back, shoulders, thighs, and arms. The skin is mottled by another crop of maculæ under the skin ("mulberry rash").

When the disease is fully developed the face is flushed, the conjunctivæ red, the pupils contracted so as to resemble pin-holes ("ferrety eye"), the tongue dry and brown, the teeth covered with sordes, the skin dry, hot, and stinging to the touch. The patient lies upon his back oblivious to all his surroundings. Headache has given place to delirium, which may be wild and fierce, but is more commonly low and muttering. There are marked ataxic symptoms—subsultus tendinum, tremors, picking at the bedclothes. Incontinence of urine and fæces sometimes occurs. The breathing is frequent, shallow, and noisy, and the pulse frequent, soft, and feeble. The macular rash now becomes petechial. The patient is in a typical "typhoid state." The stupor may gradually clear up, or, on the other hand, deepen into coma; or the patient may die from progressive weakening of the heart, with or without pulmonary complications.

In the majority of the cases which end in recovery, on or about the fourteenth day the first sign of recovery is a sound sleep, from which the patient awakes refreshed and rational. The temperature falls with great rapidity, the pulse and temperature improve; a typical crisis has occurred.

Certain *objective phenomena* of the disease require special mention. The eruption is more copious in severe than in mild cases. A dull and livid color is a grave sign. Purpura and hemorrhages are sometimes met with in bad cases. The eruption does not occur in successive crops.

The patient seems to be surrounded by a vapor of a pungent, musty *odor* which is peculiar.

The *heart* early shows the effect of the poison. The impulse is diminished, and the first sound less distinct. In grave cases with threatening heart-failure the sounds are feeble and distant, the impulse imperceptible.

The *pulse* is usually increased considerably in frequency, but may be abnormally slow (50 and even 30 per minute), which is sometimes a bad sign.

The *weak heart* and prostrate position of the patient favor congestion with œdema of the lungs. This condition is common.

Digestive symptoms have been referred to already. Vomiting, tympanites, and diarrhœa are rare, and still more so is intestinal hemorrhage.

The *urine* is scanty and high-colored. Slight albuminuria is common, and a few casts are found; but distinct nephritis is unusual. Convulsions, when they occur after the first week, are almost always uræmic and are almost invariably fatal. Some curious instances have been recorded by Stokes and Corrigan in which the convulsions were due to retention of urine.

The *duration* of the disease is from six to fifteen days; the average period is twelve or fourteen days. An abortive form is met with in

some epidemics, the disease being of a mild type and subsiding at the end of a week. It is also possible for so large a dose of the poison to be received that the patient is stricken down in a few hours or a few days. To this form the name "blasting typhus" has been appropriately given.

The most important complications are hyperpyrexia, laryngitis, bronchitis and congestion of the lungs, extreme ataxia or profound adynamia, nephritis, heart failure, and parotitis, or other inflammatory glandular swellings.

Laryngitis with œdema is a rare but very dangerous complication.

DIAGNOSIS. *Cerebro-spinal fever* is distinguished from typhus fever by greater intensity of the headache, by retraction of the head and hyperæsthesia, by greater liability to vomiting, by absence of the macular-petechial eruption of typhus and of the drunken, besotted aspect of the latter disease. In cerebro-spinal fever the patient suffers with photophobia and is liable to local palsies of the eye-muscles (strabismus) and to general convulsions. Convulsions do not occur in typhus except from a complicating nephritis or retention of urine.

Uræmia is distinguished from typhus by the preceding history, by the absence of high temperature, and the presence of œdema of the face or extremities, a history of vomiting or diarrhœa preceding the stupor. The condition of the urine and the absence of eruption are the final tests.

Pneumonia is distinguished by the frequent respiration and relatively slower pulse, and by the local physical signs and absence of eruption.

Typhoid fever is distinguished by its slow onset and marked abdominal symptoms. The eruption of typhus is petechial, and comes out on the fourth day; that of typhoid fever consists of rose spots, and appears on the seventh or eighth day.

Relapsing Fever.

An acute infectious and contagious fever, occurring in epidemics and characterized by the sudden onset of a febrile period lasting five or seven days, which is followed by an intermission lasting usually a week, and this in turn by a relapse lasting three days. Its development is favored by filth and famine, but the specific cause is believed to be the spirillum of Oberméier, which is constantly present in the blood during the febrile stage.

The stage of *incubation* lasts from five to eight days (Pepper), during which the patient may complain of malaise, lassitude, and flying pains.

The *invasion* is sudden. It is evidenced by a chill or chills, frontal headache, pains in the back and limbs, vertigo, and great physical weakness. The temperature rises very rapidly, reaching 105°, 106°, or even higher, in the first day or two. The face is flushed, epistaxis sometimes occurs, the headache and other pains persist, but delirium is not common. The appetite is usually lost, thirst intense, the tongue coated white but moist, the bowels constipated. A mild catarrhal jaundice is not infrequent. Pepper states that nausea and vomiting are

prominent symptoms, the matters vomited at times containing blood. Tenderness, with pain in the epigastrium, is frequently complained of.

The *urine* is scanty, high-colored, and frequently contains albumin and casts; when jaundice exists it contains bile pigment and sometimes blood.

There is no peculiar *eruption* in relapsing fever, but in this, as in other fevers, erythemata, petechiæ, and sudamina may be present.

The *pulse* is often very frequent and soft, and hæmic murmurs may be audible.

The *objective symptoms* are few. They consist of the flushed face, sometimes with slight jaundice and epistaxis, tenderness in the epigastrium, with moderate enlargement of the spleen and liver, and considerable cutaneous hyperæsthesia, with tenderness along the nerve trunks. Bronchitis and sometimes hypostatic congestion of the lungs, with their usual physical signs, may be present.

These symptoms continue without much change until the fifth or seventh day, when a decided *crisis* occurs. Sometimes this is deferred until the tenth day. The *temperature* within twelve hours falls from 106° or 108° to or below normal; the pulse diminishes in frequency from 120 or 130 to 60 or 70; vertigo, headache, and other pains disappear as by magic. The crisis is marked most frequently by a profuse sweat, sometimes by diarrhœa, epistaxis, metrorrhagia, or intestinal hemorrhage. The patient now enters upon convalescence without fever, and apparently makes rapid strides toward complete recovery. On the seventh day from the crisis, however, a sudden relapse occurs, with a repetition of the symptoms of the primary attack. The temperature may be higher and the febrile symptoms more severe, but the duration is shorter, lasting only three or four days. The spirilla, which disappeared in the apyretic interval, are again found in abundance. A second crisis, with its associated symptoms, now occurs. The spirilla again disappear, and in the majority of the cases there is no further bar to complete recovery. A second, third, and even a seventh relapse may occur, as in a case recorded by Pepper. Organic lesions are not usually left behind, unless they have occurred as complications; but even in ordinary cases the patient is left weak, anæmic, and with poor circulation.

Spirillum Obermeieri. Found in the blood of persons suffering from relapsing fever during the paroxysms.

Slender, flexible, spiral or wavy filaments from 16 to 40 μ by 0.1 μ. Stains with aniline colors and Löffler's blue. (See Plate I., Fig. 4, A.)

Aërobic and *motile.* Has not been cultivated on artificial media. When injected into the blood of men or monkeys produces typical relapsing fever.

The most frequent *complications* are on the side of the lungs, kidneys, and heart. Lobar pneumonia is the most frequent. The heart becomes weakened by the very high fever and thrombosis, or sudden failure results. Embolism is very frequent. Suppurative parotitis, abscess of the spleen, profuse epistaxis, abortion in pregnant women, and neuritis deserve mention.

Relapsing fever occurs at all ages, but is most common in adults.

The duration varies according to the number of paroxysms. If there is only one it is about eighteen days. Under the name "bilious typhoid" a malignant form of relapsing fever has been described. It is characterized by intensity of the symptoms of the ordinary form, and by bilious or bloody vomiting, jaundice, and delirium, or by collapse, with purple nose, a small, frequent, weak pulse, rigidity of the abdominal muscles, tenderness in the epigastrium, and cold, clammy skin. In some of the cases described by Graves, intussusception of the intestines was found after death. In other cases uræmia is an active factor.

Diagnosis. The earlier cases in an epidemic may not be recognized, unless the blood be examined, until the occurrence of the characteristic relapse. It is most likely to be mistaken for *typhus fever*, which occurs under similar conditions. The aspect of the two diseases is very different. In typhus there is a heavy, stupid, sometimes besotted expression, with slight redness of the eyes and a contracted pupil. The patient lies oblivious of his surroundings, with low muttering delirium and ataxic symptoms. In relapsing fever, on the other hand, the sensorium is rarely much disturbed, the spleen and liver are enlarged, and there is hyperæsthesia. Moreover, in typhus there is a spotted eruption, later becoming petechial. In relapsing fever this is absent.

Variola.

Variola, or smallpox, is a specific infectious and contagious fever, beginning abruptly with chill, high temperature, headache, vomiting, sweating, and intense pain in the back. On the second or third day of the disease a characteristic shot-like, papular eruption appears, the papules rapidly developing first into vesicles and then into pustules; with the appearance of the rash the temperature falls, but rises again toward the end of the week in the pustular stage (fever of maturation or suppuration). The contents of the pustules are discharged, crusts form, and are cast off about the eighteenth day. The disease may be accompanied by a number of complications, particularly hemorrhages into the skin (purpuric smallpox), and from the mucous membranes (hemorrhagic smallpox), both forms being popularly called black smallpox. For convenience of description the disease may be divided into four stages: (1) Incubation, (2) invasion, (3) eruption, (4) desquamation.

Incubation. This stage lasts from ten to fourteen days, and is usually unaccompanied by any symptoms except malaise toward its close.

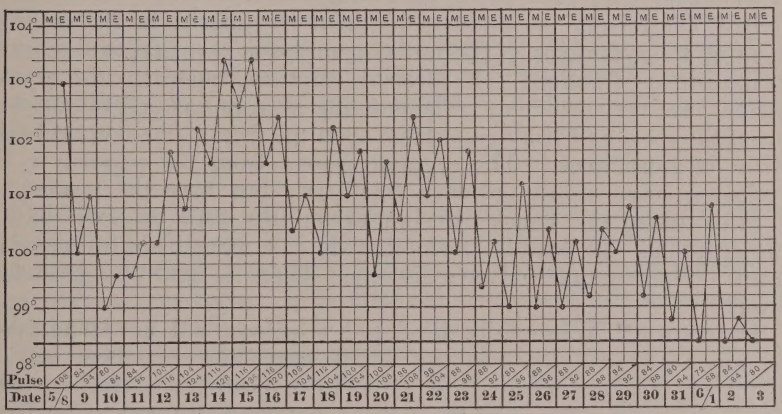
Invasion is abrupt, and is marked by chilliness or a distinct rigor, headache, severe pain in the lumbar region, and sometimes delirium or convulsions, especially in children. The most prominent symptoms are the excruciating headache and backache. The temperature usually rises rapidly to 104° F. or higher in the first twenty-four or forty-eight hours. Headache and backache continue; there is pain in the epigastrium, a coated tongue, loss of appetite, nausea or vomiting, constipation and copious perspiration. Prostration is extreme. Erythematous eruptions are not uncommon, especially on the inner surfaces of the

legs and thighs. Petechiæ also are found in Simon's triangle, whose base is at the umbilicus and apex at the knees.

The stage of *invasion* lasts generally three days; but it may be shortened to two in very severe cases or lengthened to four in very mild ones, and in complicated and hemorrhagic cases it merges into the stage of

Eruption. The characteristic eruption of smallpox appears first as minute specks resembling flea-bites. These in two or three days develop into small papules which feel like shot under the skin. In a day or two more the papules become vesicles, which at first contain a clear fluid, but which rapidly becomes turbid; they are umbilicated. In the course of another day or two the vesicles have become pustules and are globular in shape. The period of ripening or maturation, when pustulation is at its height, lasts about three days; it is characterized by a marked secondary fever, the temperature rising as high as, or higher than, in the onset of the disease. The pustules now begin to dry up (desiccation) and form dry scales or scabs which are cast off toward the end of the third week of the disease (eighteenth day); when the pustules have been deep enough to involve the true skin, characteristic scars called pits are left.

No. 140.



Temperature in smallpox. Adult: mild case.

The *eruption* appears first on the forehead, along the margin of the hair, and in the scalp, then over the rest of the face, especially about the nose and lips, subsequently progressing over the rest of the body from above downward. The eruption is most abundant upon the face and hands, often being confluent here when discrete elsewhere. The face may appear horribly swollen, bloated, and disfigured, and both face and hands are extremely painful from the great distention and the pustules, which are really small dermal abscesses.

Varieties. Three varieties of variola, depending upon the number and disposition of the pocks and upon the presence of complications, are recognized: (1) Discrete; (2) confluent; (3) malignant.

In *discrete* variola the pocks are not numerous, and are separated from each other by intervening healthy skin.

In *confluent* smallpox the pustules are close-set, occupy almost the whole body, and coalesce, so that the face looks as though covered with a black, rough mask; the mucous membranes also are covered. The symptoms of the invasion are intensified and the eruption may appear before the third day. Patients are liable to suffer with profuse salivation, uncontrollable vomiting, or diarrhœa (especially in children), and with delirium which is often violent and destructive. The face is dreadfully swollen and the eyelids may slough; the feet and limbs also may be swollen and painful. There may also be severe bronchitis and pneumonia, abscesses, extensive sloughing, and a pyæmic condition.

Malignant, or *black*, *smallpox* is a form in which the blood is so altered that hemorrhages into the skin or from the mucous membranes occur. In the former case there are petechiæ and ecchymoses upon the skin; in the latter more or less profuse hemorrhages occur from the womb, kidney, bowels, lungs, and stomach. The mind of the patient remains clear and he is conscious of his peril. The eruption is delayed or does not occur at all.

Varioloid is a mild form of smallpox occurring in a person protected, but not completely, by previous vaccination, or in a person who, from other causes, does not possess the average susceptibility. It is characterized, apart from its mildness, by great irregularity in the development of the symptoms. The initial symptoms, as a rule, are as severe as in ordinary smallpox. Prodromal eruptions, especially the erythematous, are very common. The eruption may appear first on the face, or on the chest and trunk, and later upon the face. The fever subsides with its appearance. The eruption passes from the papular to the vesicular stage, as in ordinary smallpox; but here the process, as a rule, ceases, the vesicles drying up on the fifth or sixth day of the eruption. If pustules form they do not reach their full development. The eruption is always discrete. There is usually no secondary fever.

Diagnosis. When fully developed, smallpox will not be mistaken for any other disorder. In the initial stage, however, there may be doubt whether the disease will prove to be pneumonia, cerebro-spinal meningitis, or typhus. If the patient has been exposed to smallpox and is unprotected by vaccination, and he is suddenly seized with chill, high temperature and excruciating pain in the lumbar region, there is great probability in favor of smallpox. If the patient has complained of headache, pains in the ankles and other joints, and is seized with a severe rigor, explosive vomiting, and great weakness of the limbs, the chances favor meningitis in the absence of known exposure to smallpox. In *pneumonia*, vomiting, chill, and high temperature succeed each other, but excruciating backache is wanting, and, on the other hand, the respiration is increased out of proportion to the pulse, and even in this early stage there may be cough and roughening of the respiratory murmur on one side.

Typhus fever begins abruptly with chill and high temperature; but the eruption which comes out on the third day is macular and petechial,

the temperature does not fall, the aspect of the patient is drunken and stuporous, the conjunctivæ are injected, the eye ferrety, the skin dry, hot, and biting to the touch (calor mordax).

In the papular stage of the eruption it may be mistaken for *measles;* but the red, swollen, blear-eyed, photophobic little patient with measles, with the characteristic coryza and obstinate cough, presents a very different appearance from that seen in variola. Moreover, the eruption of measles is relatively flat, smooth, and velvety; that of smallpox is acuminate, hard, and shot-like. The temperature in smallpox falls as the eruption appears; that of measles remains high and even increases. The papules of measles do not develop into vesicles.

In the vesicular stage varioloid may be mistaken for *chicken-pox.* In the latter the eruption is practically vesicular from the start, occurs without prodromata, appears first upon the chest and neck, later upon the face and scalp, is usually very scanty, and rarely becomes umbilicated or pustular. There are, however, severe forms of varicella in which fever, restlessness, and cough precede the appearance of the rash, which is copious, some of the vesicles being inflamed at the base, some umbilicated, and some with purulent contents. These cases are most common in scrofulous children whose hygienic surroundings are bad. In such cases the diagnosis cannot be made from the eruption. A consideration of the following points must decide: 1. History of exposure to varicella, on the one hand, or smallpox on the other. 2. The presence or absence of evidence of effective vaccination. 3. The age of the patient: smallpox occurs at all ages, varicella only in childhood. 4. The discovery among other neighboring children of unmistakable varicella or varioloid.

Varicella.

Chicken-pox is an acute specific infectious fever, occurring almost exclusively in children, and characterized by the appearance in successive crops of colorless or pearly vesicles, which dry up and are shed in from two to five days. It is attended with very little constitutional disturbance.

The *incubation* is generally about two weeks, but may be one or three weeks. In ordinary cases the first evidence of the invasion of the disease is the appearance of the eruption. In other cases, the severer ones, the child may be noticed for some hours or several days to be indisposed, complaining of loss of appetite, nausea, headache, and vague muscular pains. The fever is almost always moderate—100° to 101°.

The *eruption* consists of hyperæmic macules, compared by Trousseau to the rose rash of typhoid fever. These rapidly become first papules and then vesicles. The papules are not hard as in variola.

They appear first upon the chest, neck, face, and scalp, then upon the trunk and limbs. The development of the vesicles is so rapid that the eruption appears vesicular from the start. The vesicles vary in size from a pin-head to a small pea. They are very superficial, and usually rest upon a base that is slightly or not at all hyperæmic. The contents are at first watery, but subsequently become pearly. The reaction of the fluid is alkaline. Distinct umbilication is rare, and pustulation

still more rare, but both occur. Almost always the vesicles dry up and form scabs, yellowish or brownish, which drop off leaving a slightly reddened, sometimes depressed spot. Sometimes the vesicles are to be seen upon the buccal mucous membrane and upon the throat. While most of the eruption appears in the first or second day, fresh vesicles continue to appear for several days.

Desiccation usually occurs by the fourth or fifth day, and may be present in the first day or two. Often all stages, from the initial macule to the dried scales, can be seen in one case.

Usually the vesicles are widely scattered, a dozen or two over the entire body. They are most numerous upon the back, and may be as close together as in discrete variola.

In scrofulous and badly nourished children the lesions are more inflammatory and pustules are more common. If they are scratched, ulceration ensues. A gangrenous form has been described by Eustace Smith and others; the cases are apt to be fatal.

In ordinary cases during the eruption the child is rarely more than indisposed; complications are rare, and the prognosis most excellent. The physician is not often consulted except to have his opinion as to the diagnosis. (For the differential diagnosis from smallpox, see Variola.)

From *vesicular* and *pustular eczema* it is distinguished by the fever, the symmetrical grouping and discrete character of the lesions, the comparative absence of itching and burning, and its shorter course.

Impetigo is distinguished by the absence of fever, the more local character of the eruption, and the fact that it is generally pustular. It is more common upon the face and hands than is varicella.

Measles.

An acute specific infectious and highly contagious fever, characterized by coryza and bronchitis, a red papular eruption coming out on the fourth day and followed by a branny desquamation about the ninth or tenth day. The mucous membranes are especially liable to complications.

Measles occurs in epidemics, especially in cold weather, but individual cases are met with in large cities at all seasons of the year. It is so contagious that when one case develops in a household or institution almost every person exposed to it and not protected by a previous attack acquires it. Children from one to five years of age are most susceptible to the poison, but it may occur *in utero* and in old age; moreover, the same person may have several attacks, showing that one attack does not afford the same protection as in scarlatina and variola.

Measles is sometimes found in association with scarlatina and varicella, but it is especially liable to occur after pertussis.

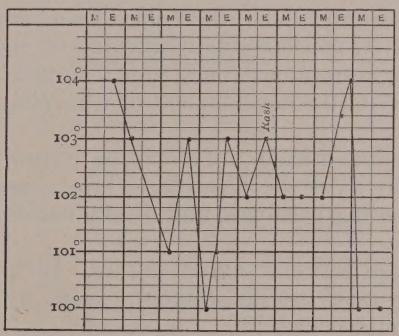
The specific cause of the disease has not yet been isolated.

The period of *incubation* lasts from eleven to fourteen days. During this time the patient may exhibit no symptoms, or may be irritable and restless, with disturbed sleep and occasional cough, and looseness of the bowels.

The *invasion* is marked by redness of the eyes and lacrymation,

sometimes with photophobia, sneezing, and an irritating, watery discharge from the nose, subsequently becoming muco-purulent, and by cough and fever. In short, the early symptoms are those of a severe coryza. These symptoms last from three to five days (generally four) before the eruption appears. But the eruption is commonly visible upon the base of the uvula and soft palate, as raised, discrete dark-red papules, several days before it appears upon the body. The *temperature* rises during the first day to 100° to 102°, or higher if the case is to be a severe one. The bowels frequently are inclined to be loose and the passages somewhat greenish. The temperature falls on the second day to normal or nearly normal, and then steadily rises until it reaches its acme with the full development of the eruption, when, in uncomplicated cases, it falls rapidly to normal. With the coming out of the eruption the coryza increases in severity, and cough is a prominent and annoying symptom. It consists of a series of five or six explosive efforts without expectoration. In several cases the cough is almost incessant, so that rest is much interfered with. It depends upon a catarrhal inflammation of the entire respiratory tract, from the nose to the bronchioles.

FIG. 141.



Measles. Temperature taken on the first day made higher as the result of school and exertion.

Objective Symptoms. The eruption on the body appears first about the neck, face, and wrists, and spreads gradually in two or three days over the entire body. It is usually most copious upon the face, which is swollen, dark-red in color, and closely set with papules, which are elevated, rounded at the summits, and feel like soft velvet to the touch. When to this picture is added that of a severe coryza with muco-serous exudate, which often glues the eyelids together and oozes out upon the face, and a corresponding condition of the nasal orifices, the physiognomy is at once seen to be peculiar. At this stage, moreover, photophobia is often considerable, the child burrowing its head in the pillows to escape light.

The *eruption* is not apt to be confluent upon the body; here the dark-red, elevated, smooth papules are very distinct. Sometimes they are grouped so as to form crescentic outlines. The eruption fades in the order in which it appeared, and is followed by a fine branny desquama-

tion. With the completion of the eruption the fever falls rapidly to or below normal, the coryza and bronchitis improve correspondingly, and in forty-eight hours convalescence is fully established.

Complications. The complications of measles affect for the most part the mucous membranes of the respiratory and digestive tracts. The bronchitis, which is always present, may become capillary, or be associated with œdema or with areas of catarrhal pneumonia. These are the most frequent and the most dangerous complications. Pneumonia may develop while the eruption is coming out, in which case the eruption is delayed or the spots have a dusky or bluish hue (black measles). More commonly, perhaps, pneumonia is discovered when, the eruption being complete, a crisis should occur.

Epistaxis is not usually dangerous. Profuse diarrhœa is very exhausting and delays the evolution of the eruption. Severe conjunctivitis, sometimes with ulceration of the cornea, is not uncommon. Otitis media occurs oftener as a sequal than as a complication. Noma, or cancrum oris, is a rare complication of measles occurring in ill-fed, badly nourished children. It is frequently fatal.

Convulsions may occur as a complication, especially when pneumonia is developing.

Sequelæ. In cases in which there has been diarrhœa, measles is sometimes followed by considerable weakening of the digestive power. The catarrh of the respiratory tract, which almost invariably accompanies it, predisposes to the development of whooping-cough and tuberculosis.

Paralysis may follow measles. It may be central or peripheral in origin, but generally is of the hemiplegic type; cases of acute poliomyelitis, acute ascending paralysis, and disseminated myelitis have also been reported.

Varieties. Measles without catarrh is rare. It cannot be recognized from a measles-like rash seen in rötheln, except by the occurrence of other cases of undoubted measles.

Measles without eruption is to be recognized by the coryza, possibly with eruption on the soft palate, the course of the temperature, and the exposure to specific infection of an unprotected person.

Black measles is the name given to malignant forms in which, owing to complications, particularly pneumonia, the skin is dusky and the eruption comes out poorly and has a bluish color. In rare instances the eruption shows a hemorrhagic tendency, the spots being livid or ecchymotic. Actual hemorrhages from mucous surfaces may occur, the patient dying in coma or convulsions.

Scarlatina.

An acute specific contagious and infectious fever, characterized by a sudden onset, with vomiting, sore-throat, and high fever, followed in twelve or twenty-four hours by a bright-red, punctiform eruption, by a very frequent pulse, by a desquamation which is often in large flakes, by a very variable degree of severity, and by a large number of complications and sequelæ, especially nephritis and inflammation of serous membranes.

Scarlet fever preferably affects children from one to five years of age. The liability to it diminishes after the tenth year; but it is very rare under the age of six months. Puerperal women are very susceptible to the poison, and the existence of open wounds favors infection.

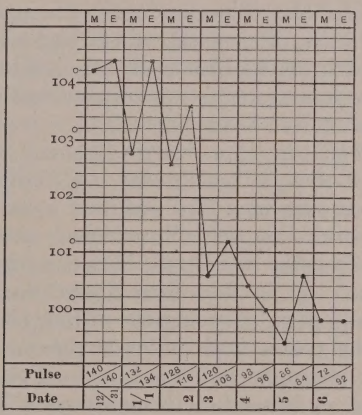
The disease occurs in epidemics at longer intervals than is true of measles. Cases are most numerous in the autumn and winter months.

The peculiar poison is doubtless a living organism, but it has not been isolated as yet. It is very tenacious of life, being capable of infecting, through clothing in which it has been retained, months after the clothing absorbed the poison.

Few diseases vary so greatly in severity in different cases and in different epidemics. It may be the mildest or the most malignant of diseases.

The period of *incubation* is remarkably short, generally from three to five days; but it may be a few hours, and in exceptional cases six days.

FIG. 142.



Scarlet fever. Mild attack; intense eruption.

The *invasion* is abrupt. It is very common to be told that a child was apparently well on going to bed, but awoke in the middle of the night, vomited profusely, and complained of sore-throat. The child is found in the morning with a temperature of 103° or 104°, a pulse of 120 to 140, and a scarlatinal eruption beginning to show upon the neck and upper part of the chest. Closer observation in such cases might have discovered that the child was feverish on going to bed, and that he had been somewhat chilly before that. Onset with decided chill, vomiting, and nervous symptoms indicates a severe case.

The *subjective symptoms* of scarlatina are few; they consist usually of pain in swallowing, with stiffness of the neck muscles, some headache, thirst, malaise, and a moderate amount of weakness. In the eruptive stage the skin itches, burns, and is frequently hyperæsthetic.

The *objective symptoms* and their order of succession are very characteristic. Vomiting is the rule, except in mild cases, and hence is of importance in diagnosis, especially in otherwise doubtful cases. The

temperature is high at the onset, frequently 103° or 104°. It falls a degree or so in the morning; but in the following evening, when the eruption is usually at its height, rises to 104° or 105°, and then gradually falls to normal in the course of a week, in ordinary cases. (Figs. 6 and 142.)

The *pulse rate* is characteristically frequent, being 120 to 160 oftener than slower. This frequency is not an indication of danger.

The *throat* exhibits a uniform flush extending over pharynx, tonsils, soft palate, and sometimes forward on the hard palate, nearly to the teeth. Sometimes darker red points can be distinguished on the soft palate. The tonsils are inflamed and project toward the median line from each side. Frequently the mouths of the follicles are blocked by a creamy-white exudate. It is not uncommon to find a severe follicular tonsillitis at the first visit.

The *tongue* is at first covered with a thick, creamy fur, through which enlarged red papillæ show. The coating soon disappears from the tip, leaving it bright red—the "strawberry tongue."

The *skin* is hot and dry. The characteristic eruption usually appears within twenty-four hours, often within six to eighteen hours, of the chilliness or vomiting which marks the onset. Sometimes it comes out very slowly, seeming to be just ready to appear, but not appearing in its full development for four or five days.

The intensity of the *eruption* varies from a scarcely perceptible erythema to the color of a boiled lobster. Usually its intensity varies with the severity of the disease. In ordinary cases the patient appears to be covered with a uniform red efflorescence; but a closer inspection shows that there are darker red spots between which the skin is more or less erythematous. It is first seen about the ears and neck, and spreads with great rapidity, covering the entire body in a day. It is most intense upon the trunk and flexor surfaces. Upon the extensor surfaces the punctate character is better seen. Pressure causes the redness to disappear, but it immediately reappears. The physiognomy of the disease is peculiar. The circle about the eyes, nose, and lips remains pale, while the rest of the face may be fiery red. Itching and burning are annoying symptoms at times.

The eruption fades gradually, in ordinary cases disappearing, except when there is pressure or irritation, toward the end of the week. Papular and vesicular forms are also seen.

It is succeeded by *desquamation*, which is extensive in proportion to the intensity of the eruption. The flakes are larger than in measles, and in severe cases the epidermis may come off in long strips. About the hands and feet this shedding is sometimes so great as to be compared to a glove. This stage may be protracted for several weeks, danger of infection lasting as long as desquamation continues.

The urine is at first scanty, high-colored, and febrile. Later, when desquamation is in progress, there is great liability to albuminuria as a complication.

Varieties. In addition to the ordinary form already described scarlatina exhibits many irregular forms. There may be only a sore-throat or follicular tonsillitis. If a rash is present it is very faint, and hence

easily overlooked. The diagnosis in such cases must be made from the fact of exposure to infection and the appearance of the throat. The occurrence of vomiting is very important in the diagnosis, as it is rare in ordinary pharyngitis and tonsillitis. Often such cases altogether escape detection until possibly a dropsy from scarlatinal nephritis indicates their nature.

Severe diarrhœa may prevent the eruption from developing upon the skin. It appears upon the fauces, and the diagnosis is based upon this, the pulse, and temperature, and the fact of exposure.

In *scarlatina anginosa* the strength of the poison is spent upon the throat. Pain is great and deglutition difficult. The tonsils are greatly swollen, so as almost to occlude the fauces, and their surfaces are covered with creamy exudate. The cervical glands are swollen and there is a tense and brawny cellulitis. Sometimes the tonsils become gangrenous and the cervical or submaxillary glands suppurate or become gangrenous, with resulting pyæmia and death. Suppuration may extend to the ears and maxillary sinus. In this form, also, a false membrane is sometimes found upon the fauces—post-scarlatinal diphtheria. It is probably not due to the bacillus of Löffler, but to a streptococcus.

In *malignant* forms the attack is ushered in with chill, followed by hyperpyrexia, convulsions, marked ataxic symptoms, or stupor. The profound blood disturbance is shown by the dusky hue of the eruption. Some patients lie in coma vigil, others are very restless and delirious. Vomiting and diarrhœa are sometimes superadded. Patients may emerge from this condition and succumb later to a nephritis or to grave anginose symptoms; but death in a few days is the rule. In rare cases the dose of poison is so enormous that death takes place in a few hours, without the appearance of any eruption.

Complications and Sequelæ. The severe local symptoms mentioned under the anginose variety, together with convulsions, hyperpyrexia, and ataxic symptoms, may properly be regarded as complications. Apart from these the most frequent are nephritis and endocarditis or pericarditis. Nephritis generally appears with the beginning of desquamation. It is nearly as frequent in mild as in severe cases, probably because the danger of exposure to cold is greater in the former, although the scarlatinal poison unquestionably has a selective affinity for the epithelium of the kidney. The symptoms do not differ from those of acute parenchymatous nephritis occurring under other circumstances. In some cases there are weakness, languor, slight fever, and prolonged convalescence; in others, œdema, anuria, convulsions or coma from uræmia. Endocarditis is often preceded by tenderness and soreness of the muscles and joints—scarlatinal rheumatism.

Endocarditis and pericarditis develop in the course of the fever, giving rise to an increase or continuance of the fever, to local pain or dyspnœa, and to the usual physical signs.

Pleuritis and meningitis also may occur. Much more common complications are otitis, peripheral neuritis, and affections of the joints, grouped as scarlatinal rheumatism. Paralyses, peripheral and central in origin, are occasional sequels of the disease. Scarlatina is found also in association with other diseases.

Diagnosis. Sudden onset, rapid rise of temperature, persistent vomiting, and sore-throat lead to suspicion of this affection. The characteristic eruption and its mode of evolution, the rapid pulse, the peculiar tongue, the circle of pallor on the face, are characteristic of the eruptive stage. The desquamation is an important diagnostic feature. Scarlet fever is distinguished from *measles* by the mode of onset, which is sudden, with chilliness, high temperature, vomiting and sore-throat, and great rapidity of the pulse; whereas the onset in measles is gradual, with coryza, cough, moderate fever, perhaps looseness of the bowels, but no sore-throat. The eruption of scarlatina occurs on the first day, that of measles on the fourth; the former consists of dark-red spots with intervening erythematous skin, the whole looking at a distance like a uniform bright-red flush; the latter consists of raised, rounded, or flattened spots or blotches, velvety to the touch, and upon the body and extremities being grouped in patches with crescentic outlines. The temperature in scarlatina subsides gradually after the rash is at its height; that of measles increases until the eruption is complete, then subsides by crisis. The rash of scarlet fever persists for six or eight days; that of measles fades as soon as it is complete on the fourth day. In the former, desquamation is in flakes or large strips; in the latter it is branny and nearly invisible. Scarlatina involves by preference the serous membranes and kidneys; measles, the mucous membranes and lungs.

Scarlatina has to be differentiated from *pharyngitis*, *tonsillitis*, and digestive disturbances attended with vomiting, high temperature, and occasionally erythematous eruptions.

In ordinary pharyngitis and tonsillitis the redness is more apt to be confined to the pharynx, tonsils, and arches of the soft palate; in scarlatina it extends as a flush over the soft and hard palate and buccal surfaces. In the former, high temperature, a very frequent pulse, and vomiting are unusual; in the latter they are the rule.

The glands of the neck also are more apt to be involved in the latter.

In *acute gastritis* there is a history usually pointing to indiscretion in eating, with constipation. The pulse is not so frequent as to suggest scarlatina, sore-throat is absent, and any erythema present lacks the characteristic dark-red points, and is not followed by desquamation.

The diagnosis from *rubella* is difficult at times. It differs from scarlatina in presenting mild catarrhal symptoms, sneezing, suffusion of the eyes, and cough, with a relatively fleeting eruption. The latter perhaps appears most frequently first upon the back and chest. Often the eruption is the first thing noticed amiss with the child. It more commonly resembles measles than scarlatina, but when it resembles the latter most it is more apt to be discrete than scarlatina and to be of a darker red. There may be a very intense rash without much constitutional disturbance, the temperature being lower and the pulse much slower than would be expected in a scarlatina presenting the same appearance. Nausea may be present, but vomiting is very rare. The post-cervical and post-auricular glands are more commonly enlarged in rubella than in a mild scarlatina, though this symptom is not invariable.

Diphtheria is distinguished by its gradual onset, patches of false

membrane developing upon the fauces early. In anginose scarlet fever, with severe follicular tonsillitis, the differential diagnosis is essentially the same as between simple follicular tonsillitis and diphtheria (which see). In addition, the pulse and temperature have a much higher range in scarlatina. The erythema of diphtheria is distinguished from the eruption of scarlatina by its fleeting character and the absence of desquamation.

Grave cases which begin with repeated vomiting, convulsions, delirium, and insomnia simulate *meningitis;* but a satisfactory cause for the latter is lacking, while the excessive heat of the skin, sore-throat, very frequent pulse, and early eruption clear up the diagnosis.

So, also, the onset with vomiting, convulsion, and high temperature resembles *pneumonia;* but in the latter the respiration is proportionately more frequent than the pulse, with altered breath and percussion sounds, while sore-throat and eruption are wanting.

Rubella.

Rubella is an acute specific contagious and infectious fever, characterized by a gradual onset with moderate fever, sore-throat, and slight coryza. The eruption, which appears without prodromata, usually resembles measles more than scarlatina. The duration, however, is shorter, the disease milder, and complications are rare.

The disease is amply proved not to be a hybrid of measles and scarlet fever. The incubation period varies from one to three weeks, but is generally about two. As a rule this period is passed without symptoms.

The invasion is without prodromata, or none more definite than languor and indisposition, the first thing noticed being the eruption. This in some cases consists of pale-red, smooth, slightly raised blotches, closely resembling measles, but more pronounced on the trunk, and discrete. This is probably a very rare form. More commonly it consists of rose-red maculæ or papules, occasionally confluent but usually discrete, and most marked upon the trunk. In still other cases the eruption closely resembles that of scarlatina, differing chiefly in being a paler red and accompanied by less heat of skin. Sometimes the eruption is circumscribed, as upon the face or limbs. It is usually the seat of considerable itching, and this may be the first symptom that attracts the patient's attention. It will be seen then that the eruption is multiform in character. Concurrently with the eruption, there is usually slight rise of temperature to 100°–101°, suffusion of the eyes, with slight lacrymation and photophobia, and slight pharyngitis; nausea is not uncommon, but vomiting is very rare. Higher temperatures have been recorded in a few cases, and so have nervous symptoms such as delirium and convulsions, but they are chiefly interesting as very exceptional possibilities. On the other hand, the disease may run its course without any fever.

The eruption extends over the body in twenty-four to thirty-six hours, less rapidly than in scarlatina, and pales much more quickly, fading on the portions of the body first attacked before reaching its

height on the last, and being completed in three or four days. Sometimes a branny desquamation succeeds.

In addition to the mild coryza and eruption, the most important objective symptom is swelling of the cervical glands, all of them sometimes being swollen, especially those behind the sterno-mastoid, the auricle, and along the margin of the hair. This adenopathy, however, cannot be relied upon exclusively in the differentiation from scarlatina and measles, as Griffith has pointed out.

Rubella has few complications: bronchitis, pneumonia, and otitis occur rarely, and still more rarely false membrane on the throat, and albuminuria. The prognosis is excellent. It ends almost invariably in recovery, except in very feeble children.

Pertussis.

Whooping-cough is a specific catarrhal inflammation of the respiratory passages, involving especially the trachea and bronchi, and characterized by paroxysms of cough, which are succeeded by spasmodic closure of the glottis and a peculiar whoop. The disease occurs especially in childhood, is contagious and infectious, and is sometimes epidemic. Whooping-cough may be conveniently divided into three periods:

1. The catarrhal stage.
2. The spasmodic stage.
3. The stage of gradual subsidence of the disease.

First Stage. The patient appears to have an ordinary cold. The amount of redness of the mucous membranes of the eyes, nose, and throat varies considerably, but there is not much discharge from the mucous surfaces. The cough is dry, and sometimes a ringing quality can be detected. The patient has slight fever, is irritable, has diminished or capricious appetite, and restless sleep. A mild bronchitis of the larger tubes can be detected by physical exploration.

The cough gradually becomes more frequent and paroxysmal, the eyes are red and suffused, and there is a muco-purulent discharge from the nose. The face often looks slightly swollen, especially about the upper part of the face and beneath the eyes.

The Second Stage. Transition from the first to the second stage is marked by the appearance of the characteristic whoop. The paroxysmal cough is made up of a series of rapid expiratory efforts, diminishing in force and duration; when these cease, there succeeds a prolonged crowing inspiration—the whoop. There may be only one paroxysm of coughing at a time, but more commonly, and always in severe cases, one paroxysm is succeeded by another. During the coughing, the child's eyes become suffused, the tears overflow, and there is a discharge of serum or muco-pus from the nose and of saliva and bronchial secretion from the mouth. The face becomes swollen and dusky. If the child is walking about it catches some object for support during the paroxysm; or if old enough, rushes for the water-closet or a basin, because the seizure usually terminates in vomiting. The matters vomited consist of tenacious mucus and the contents of the stomach. With the

mucus there may be streaks of blood, and occasionally there is pure blood. During severe paroxysms, hemorrhages are liable to occur; these are generally small and most frequently submucous. In well-marked cases, when the disease has continued some time, the face has a characteristic appearance: it is swollen, sodden, and dusky, with dull, heavy, red, and watery eyes. There is often ulceration of the lingual frænum.

The number of paroxysms varies from two or three to twenty or thirty or more in twenty-four hours, and they are worse at night.

The whoop, while characteristic, is not present in every case, being absent especially in babies and very young children. Sometimes children have "choking spells" without much coughing and without the whoop. Again, when pneumonia or measles occurs as a complication, the whoop usually ceases for the time, but may reappear later.

Third Stage. The third stage is less well defined than the first two. It may be said to begin when the nocturnal exacerbations become less frequent and severe. The number of paroxysms during the day diminishes, and vomiting is a less frequent accompaniment. Appetite begins to improve, and the child begins to gain in flesh and to pass more restful nights.

The duration of the disease is variable. Ordinarily it lasts from six to eight weeks, but it may be prolonged for several months. The patient is liable, whenever he catches a fresh cold, to a temporary return of the spasmodic cough, sometimes with the whoop.

The great majority of the cases occur before the sixth year, and most of these between the second and fourth years.

Influenza.

Influenza is a specific contagious febrile disease, occurring in widespread epidemics, having a very short period of incubation, and characterized by great prostration, marked nervous symptoms, and catarrhal inflammation of the respiratory or gastro-intestinal tracts, or both. There is great liability to relapse, and to complications, which are generally pulmonary.

The disease generally begins with the ordinary symptoms of coryza; but the headache over the eyes and root of the nose is more severe, and may be so agonizing as to mask all other symptoms. The lacrymation, rhinitis, and tormenting cough are all usually worse than in ordinary coryza. Physical weakness, weariness, and depression of spirits are almost invariably present, and they sometimes reach an extraordinary degree. Fever is usually moderate (100°–102°), but may be 103° to 104° for several days, and then gradually subside. In ordinary cases the patient seeks relief first for the headache, severe aching in back and limbs, and extreme weakness; and if these are relieved is apt to complain most of incessant racking cough, often due more to a tracheitis than to bronchitis. Nausea and vomiting are not uncommon, especially in the morning, at which time also the patient frequently feels worse than he does later in the day. Sleep is unsound and unrestful, and

may be accompanied by drenching perspirations. Severe neuralgic pains are common.

In some cases the disease attacks the stomach and bowels especially, and vomiting with diarrhœa are the prominent symptoms. In others the predominant symptoms are nervous, and great pain with prostration mask any catarrhal symptoms. Torpor and delirium may be present. Sometimes a prolonged and severe attack of asthma marks infection in susceptible persons.

Influenza has proved itself to be a disease in which considerable care is required in prognosis. To a person in ordinary good health, with proper care, it rarely proves fatal; but in the aged, or in those weakened by disease, especially of the lungs or heart, it is very grave, chiefly by causing capillary bronchitis or pneumonia, and by inducing heart failure. Less frequent complications are nephritis, otitis, cutaneous eruptions, swellings of joints, meningitis and neuritis.

The duration of the disease is from a few days to a few weeks. Convalescence is remarkably tedious, and is characterized by persistent weakness. Sweats are often annoying during this time.

The heart often continues for some time to beat too frequently and to be easily excited by exertion. Relapses are common.

Diagnosis. Influenza in the great majority of the cases is easily recognized. In certain cases, however, it has to be differentiated from *pneumonia*, *typhoid fever*, and *cerebro-spinal meningitis*.

Cases in which the disease sets in with high fever and marked chest symptoms are very apt to be mistaken for *pneumonia*. But the headache and prostration are more intense, while the respiration is not so frequent. Sweats are common, and albumin and casts in the urine are by no means rare. Physical exploration shows that both lungs are involved, though often not to the same degree. Resonance is impaired, and auscultation shows moist crepitant and subcrepitant râles, which seem to be due to an œdematous condition of the lung tissue associated with a diffuse bronchitis. A true lobar pneumonia is rarely present even as a complication.

If diarrhœa be one of the symptoms, *typhoid fever* has to be excluded. This is extremely difficult in the first two or three days. As a rule, headache, backache, nausea, and sleeplessness are at this time greater in influenza, the spleen is not so much, if at all, enlarged, the diarrhœa can be checked, and tenderness and pain in the right iliac fossa are absent.

From *cerebro-spinal meningitis* it can be distinguished by noting the fact that it begins with coryza; whereas cerebro-spinal meningitis often sets in with chill, vomiting, and faintness; the headache in the former is usually frontal, in the latter occipital and accompanied by stiffness of the back of the neck. Further, in cerebro-spinal meningitis there are often swellings of the joints, delirium alternating with coma, and in young subjects convulsions are common.

Finally, it may be said that the pronounced diagnostic feature is the preponderance of general symptoms over local inflammations. The occurrence of undue exhaustion, extreme general neuralgias and myalgias, and high fever, profuse sweats, without intense catarrh or inflam-

mation to account for them, is of the highest diagnostic significance. The presence of an epidemic, the contagious nature of the affection, the presence of the micro-organisms described by Pfeiffer, in the discharges, and the sudden onset, all point to the diagnosis of influenza.

Mumps.

Mumps, or epidemic parotitis, is an acute specific contagious disease, characterized by a sudden onset, with great swelling and pain in one or both parotid glands; by short duration, and by rapid recovery. Orchitis is liable to occur in boys over the age of puberty.

It occurs most frequently in children under ten years of age, but it may occur at any age. Males are much more liable to it than females. Life in institutions or barracks appears to render persons more susceptible. Stomatitis or sore-throat is said frequently to precede it.

The period of *incubation* is generally about two weeks, and is usually free from symptoms. The invasion is sudden, with chilliness, a rise in temperature, which is generally moderate (101° to 103°), and pain at the angle of the jaw; the corresponding parotid rapidly begins to swell, and so does the adjacent cellular tissue. The whole space between the ear and neck bulges out, the jaws are fixed, and any acid liquid, as vinegar, which stimulates salivary secretion, increases pain. At times the submaxillary glands are involved instead of the parotids. The disease may be limited to one side, or involve the opposite side as the process in the one first attacked subsides. Rarely it is bilateral from the start. When the swelling has lasted from three to five days the fever subsides, and the swelling begins to disappear rapidly. At this time, however, the opposite side may be attacked, or the testicle become inflamed. Usually it is the right testicle. In girls and women the ovary or mamma is rarely inflamed. Resolution is extremely rapid, and usually is not followed by sequelæ. Sometimes, however, deafness is left. In fact, sudden deafness sometimes announces the commencement of an attack. Atrophy of the testicle is an occasional result of the orchitis.

Cerebro-spinal Fever.

An acute specific infectious and mildly contagious disease, sporadic and epidemic, characterized by evidence of systemic infection, and generally also by symptoms depending upon inflammation of the cerebral and spinal meninges—particularly intense pain in the back and head, hyperæsthesia, retraction of head and neck, delirium, coma, and convulsions.

This disease, which is also known as epidemic cerebro-spinal meningitis and as spotted fever, is an infectious form of meningitis, probably of microbic origin. It appeared in the United States first in 1806. It was epidemic in Philadelphia from 1863 to 1865, and since then sporadic cases have been reported every year.

It is most common in cold weather and in persons under fifteen years of age. The period of incubation is unknown, but is probably short. It is free from symptoms. The invasion of the disease is abrupt,

although in some instances the patient may complaim of rheumatoid pains in the limbs or a joint, headache, and weakness. Usually the first symptom is a severe chill, which may awaken the patient from sleep. In other cases the initial symptom is a convulsion. Then quickly follow repeated vomiting, intense headache, sometimes accompanied with backache, and extreme prostration.

The rise in temperature is moderate, and the pulse is as often slow as frequent (Stillé). The face is pale and livid, expressing suffering, and the patient may toss from one side of the bed to the other, begging for some relief for his headache. The pain in the back becomes more severe, and root-pains dart in all directions, but especially into the limbs or joints, which may be swollen and tender to the touch; in fact the whole skin is hyperæsthetic and the reflexes are increased. The spinal muscles become rigid, and the head may be retracted. Less frequently the back is arched and trismus occurs. Delirium is common at night. It is often of a sportive type, the patient making absurd remarks, cracking jokes, or singing snatches of a comic song. Delirium may alternate with tonic or clonic convulsions and with stupor. The appetite is poor, the bowels constipated. A remission may occur on the third day, with temporary improvement of the symptoms.

As the attack progresses there may be strabismus, inequality of the pupils, and optic neuritis. Vertigo, tinnitus, and photophobia are common. Hyperæsthesia and delirium persist. The pulse becomes more frequent and the fever continues. In favorable cases improvement now begins, the headache and root-pains lessening, and delirium and spasms becoming less frequent. In unfavorable cases the convulsions may become more severe and end in fatal coma, or the patient may sink into a typhoid condition, with nephritis as a complication.

The skin eruptions, which explain the name "spotted fever," are not always present and exhibit no constant character. Herpes labialis and petechiæ are the most frequent; in other cases the eruption is macular or resembles that of measles.

In the malignant form of the disease death occurs in a few hours or two or three days. Such cases are apt to arise early in an epidemic. The patient has a violent chill; delirium occurs early; the headache is less intense, or at any rate gives way rapidly to stupor and coma. The pulse is frequent and feeble; there may be no rise of temperature, the skin being cool, clammy, and cyanotic. Local or general convulsions may occur. The eruption may be purpuric, and even ecchymoses occur. The urine is scanty and contains albumin and casts.

Mild cases occur usually late in epidemics. They are characterized by severe aching in the head, back, and limbs, nausea, vomiting, vertigo, and prostration. They closely resemble the nervous type of influenza, and would escape recognition except during an epidemic.

An abortive form, ending in recovery in two or three days; and an intermittent form, with exacerbations on alternate days, have been described.

The duration of the disease is from a few hours to two or three months. In ordinary favorable cases there is decided improvement toward the end of the first week, and convalescence is established in

two weeks. It may become chronic and last for weeks, and, as already stated, may be fatal in a few hours. Relapses are common in some epidemics.

The most frequent *complications* are on the part of the lungs and heart, particularly pneumonia and endocarditis or pericarditis. Pneumonia often occurs so early that it is difficult to decide whether it is primary with marked nervous symptoms, or is only a complication of the cerebro-spinal fever. Nephritis also occurs.

The most frequent *sequels* are deafness, blindness, headache, and local palsies.

Diphtheria.

An acute specific infectious and contagious disease, sporadic and epidemic, occurring especially in children from one to six years of age, and characterized by insidious or abrupt onset, with moderate fever, and the development upon the fauces or upon any abraded surface of a grayish-white false membrane, which has a tendency to extend, especially to the larynx. The subsequent phenomena are those of stenosis of the larynx, toxæmia, with or without superadded uræmia or marked cardiac weakness; it is further characterized by the liability to paralysis as a sequel.

Diphtheria is spread by inhaling the expired breath of a diphtheritic patient, or breathing air which has been contaminated by the clothing of the patient or the discharges from his nose and throat. It may also be transmitted directly, as when a fragment of membrane is ejected by coughing and infects the mouth or eye of physician or attendant. Moreover, it is contained in the sewers of large cities where the disease is endemic, and it persists in damp cellars if they have once been infected. Hence sewer gas and cellar air may carry the disease. There is reason also for believing that a similar disease affects birds, fowls, and cats at times, and from them may be transmitted to man.

The specific poison is the Klebs-Löffler bacillus and its toxin.

While children from one to six years of age are especially liable to it, no age is exempt—neither the newborn babe nor the very aged.

One attack does not protect a person completely against a subsequent attack.

The period of *incubation* varies from a few days to two weeks, or perhaps longer in exceptional cases. As a rule it is less than a week. It is shorter when the poison is virulent, and when infection has been upon abraded surfaces.

The *onset* in mild cases is deceptively free from positive symptoms. The child is languid, perhaps slightly chilly, and has a little fever, with thirst, impaired appetite, and discomfort in swallowing. Unless the nature of the trouble is suspected the child is not thought ill enough to be kept indoors. The throat is slightly inflamed, especially about the tonsils. The child may protest that there is no pain on swallowing. In from twelve to twenty-four hours from the onset, sometimes later, a grayish pellicle will be found upon the tonsils, and the cervical glands be swollen.

In more severe cases the onset is with chill or chilliness, followed by

a rise in temperature to 102° to 104°, sore-throat, and sometimes vomiting, though this is not so common as in scarlatina. Convulsions and delirium may occur if the fever be high or the case malignant, but they are not common. Disgust for food makes it difficult to nourish the patient. Headache, thirst, and aching in the back and limbs may be complained of. Prostration is often very pronounced from the first.

Objective Symptoms. As pointed out by Buzzard and McDonnell, the patellar tendon reflexes are often abolished as early as the first day. The characteristic false membrane appears first as a grayish pellicle upon one or both tonsils, and spreads thence to the soft palate and pharynx. The membrane soon becomes thicker and whitish in color; when fully developed it appears like white or grayish-white parchment, not lying loosely upon the surface, but imbedded in the mucous membrane, the inflamed swollen edges of which rise above the false membrane, surrounding it "as the crystal of a watch is surrounded by the rim" (J. Lewis Smith[1]). As the membrane becomes older it may be brownish, or even blackish in color, if tincture of iron has been given. If it is forcibly torn from the underlying surface hemorrhage is excited and the membrane is re-formed. As the membrane loosens spontaneously there is often marked inflammatory reaction at the edges of the surrounding mucous membrane, and in the tonsils there may be decided sloughing with a dark, gangrenous appearance.

The temperature usually falls by the second or third day, but this does not indicate either a favorable or an unfavorable end. A temperature but little above normal is not uncommon in profound toxæmia.

Albumin is usually present early, and often tube-casts and renal epithelium also can be found. The submaxillary and cervical glands are swollen and it may be difficult to open the mouth sufficiently to inspect the throat.

In *favorable cases* the membrane ceases to extend after three or four days; there is no extension to the larynx; the urine is free from albumin, or only slightly albuminous, and the pulse 100 to 120 and of good force.

In *unfavorable cases* the membrane shows a tendency to extend, either upward into the nasal fossæ, producing a thin, irritating, excoriating discharge from the nostrils, and rendering mouth-breathing necessary. It may extend also to the ears through the Eustachian tube, or into the maxillary sinus. Or the extension may be downward into the larynx, producing laryngeal stenosis. This is announced by hoarseness, with rapidly increasing difficulty in breathing. Inspiration is high-pitched, noisy, and difficult; the patient brings all the accessory muscles of respiration into play, the alæ of the nose play, the ribs are sucked in, and still he pants for breath. Every now and then a paroxysm of coughing produces cyanosis.

In other unfavorable cases the throat symptoms are not dangerous, but uræmia develops. The urine is scanty, contains a large amount of albumin, considerable blood, and numerous blood, epithelial, and granular casts. There is œdema of the feet and puffiness of the eyelids.

[1] Keating's Cyclopædia of Diseases of Children, 1889, vol. i. 606.

There is apt to be repeated vomiting; convulsions followed by coma and death may end the scene, or the patient may slowly emerge from the dark valley.

In still other cases the diphtheritic poison affects the heart. The pulse becomes feeble and very frequent, the first sound very faint; acute dilatation of the right heart may occur. There may be faintness and a tendency to cyanosis on the slightest provocation, or attacks of sinking and faintness may come without warning; in still other cases sudden exertion induces paralysis of the heart, and death.

In some malignant cases the patient is overwhelmed by a large dose of the poison, and dies in from one to three days in collapse from acute toxæmia, without there being any special local symptoms to account for it. In others the false membrane extends rapidly over the fauces, pharynx, and nasal cavities to the larynx; death occurs early from obstruction, or if postponed there is extensive sloughing, with death from secondary blood poisoning or septic pneumonia.

In exceptional cases the membrane is primary in the nares or larynx or develops upon some abraded surface, as a burn, or in the vagina of a puerperal woman. It may also attack the mucous membrane of the eye or the seat of a recent operation. Diphtheria also occurs as a complication of other diseases, particularly scarlet fever.

The most frequent sequelæ are anæmia, albuminuria, and paralysis. The latter comes on in from one to two weeks after convalescence has set in, but it may appear much earlier, and in exceptional cases later. It may be marked simply by loss of the knee-jerk, which has been alluded to already in the symptomatology, or involve the palatal and pharyngeal muscles, causing nasal voice, difficulty in swallowing, and regurgitation of food through the nose, or there may be multiple peripheral neuritis.

The Pseudo-Diphtheritic Bacillus resembles the genuine in all respects, except that it is not pathogenic. It seems to be an attenuated form of the former.

Löffler's or the Klebs-Löffler Bacillus. This is found in diphtheritic pseudo-membranes, especially in the deeper portions. It is not found in the blood.

Morphology. A bacillus 2 to 3 μ long by 0.5 to 0.8 μ broad, straight or slightly curved, with very many irregular forms.

Biological Properties. It is facultative anaërobic, non-motile, and does not liquefy gelatin. It multiplies by fission. Stains with Löffler's blue. Certain points are stained intensely, almost black. It grows in nutrient gelatin, nutrient agar, or bouillon, but best of all in Löffler's blood-serum mixture (see page 156) at 35°. (Death-point, 58°; ten minutes' exposure.) It forms large round elevated colonies, grayish-white in color and moist. There is no visible growth on potato. Milk is a good soil. (See Plate II., Fig. 4.)

On *inoculation* it causes a diphtheritic pseudo-membranous inflammation.

It generates a very poisonous toxin.

Diagnosis. Diphtheria is distinguished from ordinary *pharyngitis* by the presence of membrane. From *follicular tonsillitis* by the pro-

jecting mouths of the follicles containing a creamy white exudate. Later the exudate may cover the entire surface of each tonsil and be difficult to distinguish from false membrane. The points of distinction are that in the former the exudate lies upon the surface and can be brushed off without force and without leaving a bleeding surface; whereas in diphtheria the membrane is imbedded in the mucous membrane and cannot be torn from it without force. A raised, red, inflammatory border of mucous membrane at the junction of the patch is strongly suggestive of diphtheria. In tonsillitis there is no appearance of membrane upon the soft palate or pharynx. Furthermore, in tonsillitis the onset is attended with more fever and pain in swallowing than in simple tonsillar diphtheria. The existence of albuminuria and swelling of the cervical glands indicates diphtheria, and the absence of knee-jerk is an important diagnostic sign of diphtheria.

Erysipelas.

An acute specific contagious and infectious disease, characterized by a sudden onset, with a bright-red eruption, usually starting upon the face near the nose or mouth, and tending to march, with raised border, over the entire face and invade the scalp. It is attended with burning heat of the skin and great disfigurement from swelling.

The specific cause of erysipelas is the streptococcus erysipelatosus. It is carried to a slight extent by the air, and still more in the discharges, especially those of the nose. Repeated attacks occur in persons with chronic naso-pharyngeal catarrh, carious teeth, or a sinus. It is liable to attack persons with open wounds (surgical erysipelas), and puerperal women, producing in these cases sloughing and septicæmia. When on the body it spreads over a greater extent than when primary on the face, hence its name, "the red runner." It may pass from the heel to the thigh, and over the trunk, lasting for weeks. One attack does not protect against another, but in case there is any focus in which the streptococci linger it actually predisposes to another.

The period of incubation is usually from three days to a week. Preceding the invasion there can usually be had on close inquiry in facial erysipelas a history of sore-throat and some enlargement of the cervical lymphatics. The invasion is sudden and is marked by chill. The temperature rises rapidly to 104° or 105°, and in the next two or three days may rise still higher. Coincidently with the rise in temperature the portion of skin to be affected burns, tingles, is tender to the touch, and may be seen to be reddened. The redness increases in intensity and extent, while the skin is swollen and slightly œdematous. The part of the face to be affected is usually the cheek in close proximity to the nose, less frequently near the mouth and ear. The affected part is tender and the seat of burning or smarting pain. Vesicles and blebs often form when the inflammation is very intense. The redness disappears upon pressure, but quickly returns; sometimes it has a dusky, purplish hue. A marked characteristic of the disease is its tendency to spread. In ordinary cases it involves one cheek, eyelid, and ear, and travels across the bridge of the nose to the other side. The inflamma-

tion is most intense when it is spreading; the advancing margin is raised, tense, and brawny; the line is thus sharply drawn between healthy and inflamed tissue. The loose tissue about the eyes swells enormously, both eyes are closed, the entire face swollen, red, and with vesicles and blebs here and there. Curiously the chin escapes. The redness and swelling begin to subside in the part first attacked before the process has reached its height on the opposite side. As a rule, facial erysipelas does not extend beyond the face, the scalp and neck being spared. The scalp, however, is more frequently affected than the neck; occasionally it results in extensive cellulitis of the scalp, with the production of a septic constitutional condition and much local sloughing. The submaxillary glands are more or less enlarged, sometimes so much as to prevent the taking of solid food. While the erysipelas is extending the fever continues and is sometimes alarmingly high. The pulse is frequent and soft. Nocturnal delirium is not uncommon in severe cases, and sometimes nausea and vomiting are frequent. The bowels are usually constipated. The urine is high-colored, frequently contains a small amount of albumin, and actual nephritis is liable to occur.

In favorable cases of facial erysipelas the process is at an end in a week or less. It may be prolonged to two weeks, subsiding by crisis or lysis, and convalescence is usually rapid. The vesicles or bullæ dry up into yellowish crusts and the epiderm is shed in large or small pieces according to the intensity of the process.

Pneumonia and nephritis are the most frequent complications. Meningitis, pericarditis, and endocarditis also occur. Erysipelas may extend inward and involve the fauces, pharynx, and larynx, producing œdema and death from suffocation.

Sequelæ. If the scalp has been involved, falling of the hair occurs. The cervical adenitis may result in abscess; chronic nephritis may result. Otitis media occurs occasionally, and so do keratitis and abscess of the eyelids.

On the other hand, erysipelas is credited with causing the disappearance of lupus, chronic eczema, and sarcomata.

Diagnosis. *Herpes zoster* of the face and forehead is distinguished from erysipelas by the fact that vesicles appear first, followed by erythematous redness, and that they are limited by the median line, and are preceded and accompanied by sharp neuralgic pain, whereas erysipelas affects both sides of the face, and vesicles appear at the height of the disease; the pain is much less in erysipelas.

From *dermatitis* of various kinds it is distinguished mainly by the sharper febrile reaction, the raised border of the eruption, which begins first on one side and spreads to the other. Erysipelas is rarely equally intense upon the two sides. Dermatitis frequently is. The latter exhibits often a rough surface, whereas until vesicles appear erysipelas is smooth and shiny.

Chronic *erythematous eczema* occurs in middle-aged and old persons, is afebrile, accompanied by little swelling but a great deal of itching, and runs a slow course.

Cholera.

An acute specific infectious disease, endemic in parts of India, but occurring in epidemics elsewhere, characterized by the outpouring into the stomach and bowels of large quantities of a serous fluid resembling rice-water, which fluid is usually vomited and discharged from the intestines. It is further characterized by an algid state of collapse and by painful muscular cramps.

The specific poison of cholera is believed to be the comma bacillus of Koch, and its ptomaïne.

The native habitat of cholera is India, particularly in the neighborhood of Calcutta; here it is endemic and thence it is liable to spread in successive epidemic waves along the lines of travel by sea and land, over the whole world. It is scarcely, if at all, contagious; the poison is contained in the vomit and dejections, which contaminate the drinking-water, food, and clothing. It preserves its vitality for long periods of time in water, especially if slightly alkaline and containing vegetable matter, and in moist clothing, as rags.

The period of *incubation* is probably short in the majority of cases, lasting only a few days. Occasionally it is two weeks. There are usually no definite symptoms during this time, but there may be a sense of weakness, with loss of appetite and dyspeptic symptoms.

First Stage. The first stage, that of premonitory diarrhœa, is better regarded as the beginning of true cholera. It is characterized by profuse watery stools of a yellow or light-yellow color, and alkaline in reaction. They are accompanied by a rumbling noise in the bowels, but are passed without pain. From six to a dozen of these passages occur in twenty-four hours. The patient feels faint and exhausted after them, and may suffer with nausea, but vomiting is not usual. In severe cases there may be cramps in the calves of the legs. The voice is faint and husky, thirst intense, the tongue white and moist. The temperature is normal or slightly depressed.

This stage may last from two days to a week, depending upon treatment. In some cases it is wholly absent, and the patient is ushered abruptly into the

Second Stage. This usually comes on during the night. The patient is seized with vomiting which is at first bilious, but the fluids rapidly lose all color and become like rice-water. The stools likewise resemble water in which meal has been stirred, or in which rice has been soaked—a semi-transparent fluid with particles of epithelium resembling rice floating in it. This fluid seems to well up and regurgitate rather than to be vomited from the stomach, and to gush in quantities of a quart or two from the anus. Sometimes vomiting and diarrhœa occur at once. The patient has unquenchable thirst, and is tortured with painful cramps of the toes, legs, belly, and diaphragm. As the discharges continue the patient becomes more and more exhausted; the nose is pinched and twisted, the eyes sunken, the lips bluish, and the whole body may shrink beyond recognizable proportions.

The skin is cold and moist, the breath icy, and the temperature

under the tongue is sometimes as low as 78° to 80° F. In the vagina and rectum it may be normal or slightly above normal. The patient, however, often has a sensation of heat. The urine is very scanty, containing albumin and sugar, or it may be suppressed. The pulse is very small and feeble, 100 to 120. The mind is clear, but the patient is listless, answering questions in an extremely faint voice and with manifest effort.

Third Stage. From this collapsed and algid condition the patient may slowly emerge, the skin becoming less cold, the cramps less severe. A return of the secretion of urine is a hopeful sign. The reaction, however, may simply introduce a low typhoid condition, with fever, dry brown tongue, subsultus, low muttering delirium, and coma.

In some cases serum is poured out into the stomach and intestines and is retained there. The patient may be seized while walking with dizziness, faintness, extreme prostration, and early collapse.

In other cases the patient is smitten down with profuse vomiting and purging, dying algid and collapsed in a few hours, no reaction appearing.

In favorable cases the vomiting ceases, the stools become less frequent and are tinged with bile and have a fæcal odor. The urine increases in volume, while the albumin diminishes. Convalescence is very protracted. Anæmia, great dibility, feeble digestion, and sometimes obstinate diarrhœa delay complete recovery. Relapses are liable to occur.

In other cases reaction brings improvement in the gastro-intestinal symptoms, but uræmia develops, death following in convulsions or coma.

The most frequent *complications and sequelæ* are eruptions, chiefly erythematous, ulcerations and bedsores, parotitis, and a painful tetanic spasm of the flexor muscles of the hands, forearms, legs, and feet, occurring between the tenth and fifteenth days of convalescence (Stillé).

Diagnosis. The chief points in the diagnosis from other affections are the knowledge of exposure to cholera; the character of the vomit and dejecta, which contain the comma bacillus (for its detection see under Bacteriology); the cyanosis; the rapid development of collapse, with cold skin, icy breath, torturing cramps, and greatly shrunken visage and body.

Cholera morbus differs in that the stools remain turbid with bile or fæcal matter, or contain blood; they never present the rice-water appearance. Moreover, the passages are frequently preceded by colicky pains. Cyanosis and collapse are extremely rare. The stools do not contain the cholera bacillus.

Other forms of acute *toxic gastro-enteritis*, whether from ptomaïne poisoning or from a corrosive poison, are to be distinguished by the history, the difference in the character of the stools, and the comparative absence of painful cramps in the legs, of cyanosis, and of collapse.

Bacteriological Diagnosis of Cholera. Koch remarks:[1] As cholera resembles in clinical symptoms cholera nostras, infantile cholera, certain forms of peritonitis, certain organic poisons, and poisoning by arsenic, it is important to attain some means of making a definite diagnosis.

[1] Zeitschrift für Hygiene und Infektionskrankheiten, 1893, vol. xiv., No. 2.

The Microscopical Examination. Cover-glass preparations of the dejections of the patient or of a flake of mucus from some fluid of the body are made. The preparation is stained by Ziehl's red (fuchsin). In addition to the cholera bacilli, the bacillus coli communis and other intestinal bacteria are found. The cholera bacilli lie in groups in the thread-like strands of mucus. They form in heaps, the bacilli lying in the same direction. Koch holds that this mode of grouping is characteristic and diagnostic. He further holds that if bacilli coli are in close proximity to numerous scattered bacteria resembling the cholera bacilli, the case is one of Asiatic cholera.

Peptone Cultivation. A small quantity of the dejection of some flakes of mucus is inserted with a platinum loop into a sterilized 1 per cent. peptone solution. The solution is maintained at 37° C. The cholera bacteria are aërobic, and develop on the surface of the peptone, while the fæcal bacteria remain in the deeper layers. As soon as the peptone is cloudy a drop from the surface is examined microscopically. Within six hours the surface is overwhelmed with a pure culture of cholera. Later they are mixed with bacteria coli. The examination should be made from six to twelve hours after the peptone solution is inoculated. The peptone solution should be strongly alkaline, and a 1 per cent. solution of common salt added. Care must be taken to see that the solution contains sufficient soda. In plate cultivations the cholera bacilli are overwhelmed by the fæcal bacteria.

Gelatin Plate Cultivation. Three dilutions are prepared and poured into double-bottomed vessels. The vessels must be submitted to a temperature which is warm, but does not liquefy the gelatin, as about 22° C. The colonies are seen in from fifteen to twenty hours. If the gelatin becomes liquid the cholera colonies resemble Finkler's bacteria.

Agar Plate Cultivation. The growth is not so characteristic as it is in gelatin. The cholera bacilli form large colonies of a light gray-brown transparent appearance. Colonies of other bacteria are less transparent. The colonies can be obtained in from eight to ten hours after exposure to a temperature of 37° C. Microscopical examination of the colonies must be made.

Cholera-red Reaction. Cholera cultivations contain indol and nitrous acid, and produce a red color if sulphuric acid is added. This color is produced by other bacteria also, but by none other of the bacteria that are curved. Care must be taken to make the cultivations with suitable peptone, and to have the sulphuric acid free from nitrous acid.

Experiments on Animals. The agar cultivations are employed. They must be introduced into the abdominal cavity of the guinea-pig. The injection must not be made into the intestine, a matter which requires considerable practice. No other spirillum or curved bacillus produces the symptoms.

Dengue.

An acute specific contagious disease, occurring in epidemics and characterized by severe pains in the head, back, and joints, various skin eruptions, a prolonged convalescence, and a very low rate of mortality.

The disease occurs in epidemics in tropical and subtropical countries,

and rarely in cooler climates. It derives its name, dengue (dandy), from the stiff and unnatural gait assumed by patients in convalescence. In the southern parts of the United States an expressive name given to the disease is "breakbone fever."

The specific cause of the disease is believed by Dr. McLaughlin to be a micrococcus which he isolated. The period of incubation is short, varying, however, from a few minutes to several days, or even a week. Invasion is very sudden and is rarely preceded by any prodromata. It is marked by chilliness or a chill, and very severe pains in the head, back, and limbs. In children the onset may be by convulsions, which are sometimes followed by stupor and vomiting. The pains are sometimes excruciating and are accompanied by tenderness of the muscles; there is extreme debility. The temperature rises to 102° or 103,° but rarely is much higher.

The pulse is frequent—110, 120 or more. In from one to three or five days the temperature falls to or below normal (the remission), accompanied by sweating or diarrhœa, and fluctuates about this level for several days, when a second and moderate rise in temperature, which is of short duration, occurs. During the first rise in temperature there is a transient, generally scarlatiniform, rash, which is not followed by desquamation. The urine is febrile, but not albuminous. During the remission eruptions—scarlatiniform, herpetic, urticarial, or like miliaria—begin to appear, accompanied by the secondary rise of temperature. The eruptions may be in successive crops and are followed by desquamation. Convalescence is now established, but may be interrupted by relapses. The most frequent complications affect the nervous system, but bronchitis and diarrhœa occasionally occur.

Malarial Fevers.

A group of fevers associated with the protozoan organism of Laveran, and characterized by periodic paroxysms of chill, fever, and sweat. They are not contagious, but can be transmitted by inoculation.

Malarial fevers, while most prevalent in tropical and subtropical regions, are found also throughout the temperate zone, especially in autumn and spring. In Europe their favorite habitat is Italy, and in the United States the southern and southwestern States. Conditions that especially favor their development are marshes and swamps, fed partly by sea-water; low ground along streams of slow current; and freshly upturned soil.

The poison is carried in the air, hence winds blowing from marshes or other infected districts are especially dangerous.

The specific poison in malarial fevers is no doubt organic. The protozoan organism described by Laveran exhibits several different forms, which he regards as stages in the development of one organism, but which may be different species. Golgi maintains that there are several distinct varieties of parasites whose periodicity in development and sporulation corresponds with the different types of fevers.

Intermittent Fever. This is a type of malarial fever in which the temperature remains normal between the paroxysms. A malarial

paroxysm is characterized by (1) chill, (2) fever, and (3) sweating, occurring in the order named and in immediate succession. The time between the beginning of one paroxysm and the beginning of the next is called the "interval," that between the conclusion of a paroxysm and the beginning of the next the "intermission." The interval varies in different forms of intermittent fever: in the quotidian there is a paroxysm every day, with an interval of twenty-four hours; in tertian there is a paroxysm on alternate days, with an interval of forty-eight hours; in the quartan there is a paroxysm every third day, with an interval of seventy-two hours. In double quotidian there are two paroxysms in the twenty-four hours, but not of the same intensity.

In the double tertian there is a paroxysm every day, the first and third and second and fourth corresponding as to hour and intensity. That is to say, if there be a severe paroxysm at 10 A.M. Monday, there will be another severe paroxysm at 10 A.M. Wednesday, while on Tuesday and Thursday there will be milder paroxysms, but at another hour than 10 A.M.

In the double quartan severe and mild paroxysms succeed each other every other day, but each third day is free from any paroxysm.

While the rule is for malarial fevers to occur periodically at the same hour, the second paroxysm may occur an hour or two earlier (anticipation) if the disease is growing worse, or an hour or two later (postponement) if it is growing better.

Quotidian intermittents are slightly more common than tertian, while the quartan variety is rare.

The *incubation* period probably varies widely, depending upon the intensity of the poison. As a rule repeated exposure is necessary to develop the disease in temperate climates. During this period the patient may suffer with headache, drowsiness, pains and aching in the limbs and back, constipation, a coated tongue, and thirst.

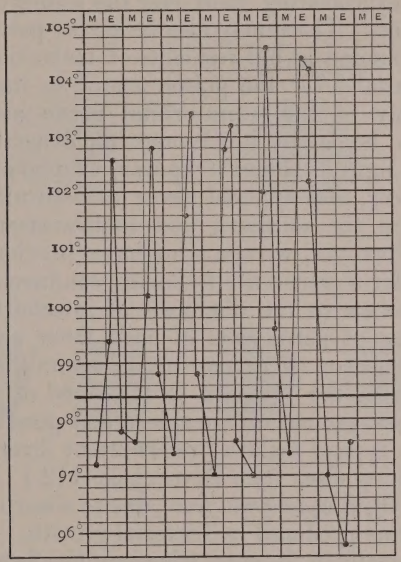
The *onset* of a typical malarial paroxysm is marked by chilly sensations, especially along the spine, accompanied by yawning and the development of "goose-flesh." Then a decided chill sets in, the patient shaking violently. The face is pale and pinched, the lips blue, the nose pointed; as the chill becomes worse the teeth chatter, the whole body feels cold, the skin feeling rough, dry, cold, and harsh. The finger-nails and toe-nails are blue, the skin being wrinkled upon the palmar and plantar surfaces. The superficial bloodvessels are so contracted that a drop of blood is obtained with difficulty. The voice is thin and weak, almost inaudible.

The volume of blood driven from the surface leads to congestion of the viscera, particularly the spleen, liver, and stomach. Nausea and vomiting are not uncommon. The spleen is perceptibly enlarged, and frequently the liver is also.

Although the surface temperature is depressed, the internal *temperature* is rising, and may be two or three degrees above normal. By degrees the severity of the chill abates and the patient asks to have the extra bed-clothing removed. Reaction has set in. The surface bloodvessels dilate and the skin becomes flushed. The temperature continues to rise, often reaching 103° to 106°, pulse and respiration increasing

correspondingly in frequency. The patient complains of a throbbing, dizzy headache, and vomiting may recur. The bowels remain constipated. The temperature now begins to fall, and the sweating stage succeeds. Perspiration appears first upon the forehead, face, and neck, and by degrees extends over the rest of the body. The perspiration becomes more and more profuse, until the whole body is drenched with it. All the subjective symptoms vanish with wonderful rapidity, and the patient, with the exception of exhaustion, seems to be restored to complete health. The hot stage lasts from one to two hours, the cold stage from three to eight hours, and the sweating stage from two to six hours.

FIG. 143.



Intermittent fever. Temperature every six hours. Morning and evening temperature and highest at chill.

In the interval between paroxysms the patient is free from fever, but is anæmic, weak, and has impaired appetite, and constipation.

During the entire paroxysm the mind remains clear.

The chief *objective symptom*, apart from the phenomena of chill, fever, and sweat already described, is the occurrence of plasmodia in the blood (see under Blood).

IRREGULAR FORMS. *Irregular forms* of intermittent fever are more common in Philadelphia than the typical form just described.

In the *mild form* the patient complains of great lassitude, irritability of temper, and drowsiness during the day, but at night tosses upon his bed and gets up in the morning more tired than when he went to bed. The back and limbs ache, and feel as though they would give way under him. There is severe throbbing headache, with some dizziness and faintness. The bowels are constipated, the tongue heavily

coated with yellow fur. The temperature is moderately elevated and the patient has great thirst. Nausea and vomiting are absent, though there is little desire for food. There may be a burning feeling referred to the splenic region. The patient is worse on alternate days, and the attacks may be preceded by slight creeping chills. On inquiry the patient will be found to live in a low-lying district near one of the rivers, or in a damp house over an unclean, moist cellar, or adjoining a place where fresh soil has been upturned.

In the form known as "*dumb ague*" there is periodically great depression, with aching in head and limbs, a sensation of coldness rather than chilliness, but no marked fever and sweating. Nausea and vomiting may, however, be present. Da Costa says he has seen it manifest itself by excruciating pain over the kidney, and almost entire suppression of urine. There may also be severe paroxysms of gastralgia. It is more common in old residents of malarious districts.

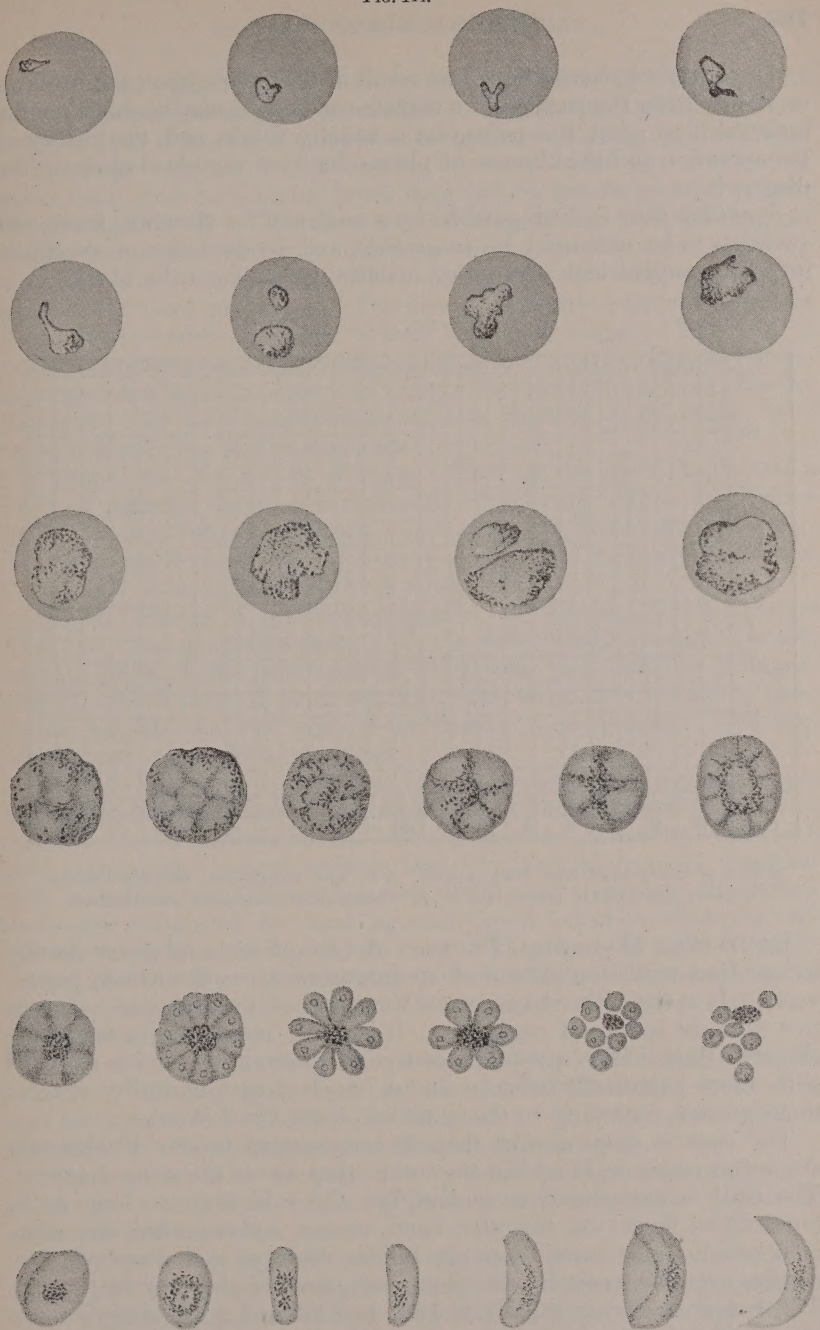
In *masked* malarial fever the poison manifests itself in an attack of neuralgia, especially of the supra-orbital nerve and gastric nerves. Malaria may also be latent until some impairment of the resisting-power brings it to light. Hence it appears as a complication of pneumonia and dysentery, and typhoid fever (constituting typho-malarial fever), especially in the southern and southwestern portions of the United States. Moreover, women who have previously had intermittent fever may suffer a recurrence following confinement.

The essential points in the diagnosis of intermittent fever are the periodical recurrence of paroxysms of chill, fever, and sweating, or of attacks of dumb ague, or of paroxysms of neuralgia, without organic lesion, associated with the presence in the blood of pigment and plasmodia, and with enlargement of the spleen and possibly of the liver.

Diagnosis. A typical malarial intermittent fever is not likely to be mistaken for anything else. (See Fever, page 112.) It needs, however, to be distinguished from *septicæmic fever*, due to absorption into the blood of pus and the toxins produced by bacterial growth. Such fever occurs in *tuberculosis*, especially in the stage when cavities form and pus collects; in the puerperal state, in empyema, subphrenic abscess, abscess of the liver, or it may occur in any form of suppuration. Here also, then, are recurring chills, with fever and sweating, but the attacks are not regularly periodical and intermittent; sometimes the fever is intermittent and sometimes remittent, the chills recur at irregular intervals, and are not so violent as in the malarial attack. The essential difference, however, lies in the fact that a local cause can be found to explain them, either tuberculosis of the lung or some other viscus, or a collection of pus in an organ or cavity, or a fœtid discharge from the womb, with local tenderness or peritonitis; moreover, the patient loses flesh more or less rapidly, his blood is free from malarial germs and pigment, and quinine does not control the fever.

From the intermittent fever of *hepatic* origin (described elsewhere by the author) the diagnosis is more difficult, in that physical signs of any local trouble may be wanting. But the fever is not regularly intermittent, is not controlled by quinine, but may be by measures directed to the origin of the trouble, and jaundice may be present.

FIG. 144.

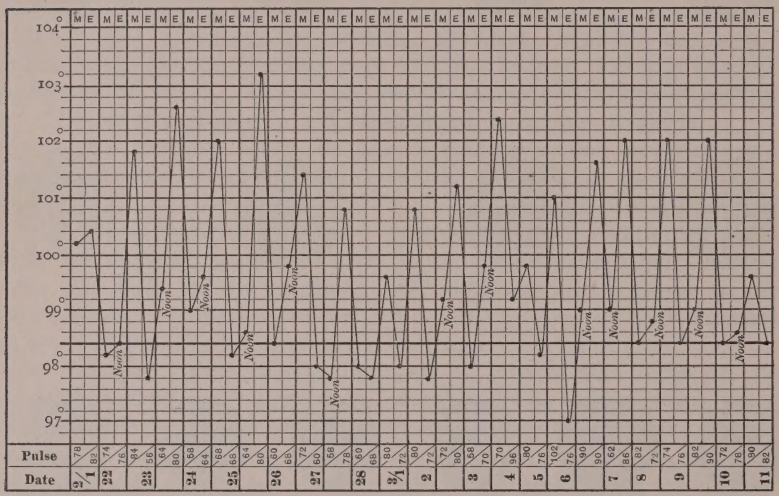


The first twelve figures show the malaria plasmodium. It is a pale amœboid body inside the red corpuscle. It increases in size at the expense of the corpuscle. In the last four of the twelve it is enlarged and contains pigment granules derived from the hæmoglobin. The figures of the fourth row show progressive stages in the process of cleavage of the plasmodium and shifting of the pigment granules. In the fifth row the process of cleavage is seen to be completed, and final isolation of the spores has taken place. The dark granules are pigment granules. The last row shows oval parasites.—Laveran's corpuscles observed in atypical cases of malaria. (From GOLGI, "Studien über Malaria," *Fortschritte der Medicin*, Bd. iv., Tafel III.)

Urethral fever, occurring as the result of operations upon the urethra, or simply from the passage of a catheter or bougie, may be mistaken for malarial fever; but the paroxysm is usually single, and the history of the operation and the absence of plasmodia from the blood clear up the diagnosis.

Syphilitic fever is distinguished by a tendency for the chill, fever, and sweating to be nocturnal in recurrence, and by evidence of syphilitic infection coupled with absence of malarial germs from the blood.

FIG. 145.



A form of intermittent fever from syphilis. J. D., aged twenty-six. Secondary period. Mercury and iodide of potash relieved it. Observe that the pulse is not increased.

REMITTENT MALARIAL FEVER. A type of malarial fever characterized by a remission instead of an intermission in the febrile paroxysms. It is due either to a greater intensity of the malarial poison or to a different species of organism. It is much more rare in temperate climates than either quotidian or tertian intermittent and is attended with more gastric disturbance and a much larger mortality (twelve times greater, according to the statistics of the Civil War).

The *onset* is more abrupt than in intermittent fever. Prodromata are not so common, but when they occur they are of the same character. The chill is not usually so violent, nor the cold stage so long as in intermittent fever; on the other hand, nausea and vomiting are common, and in some cases there are bilious vomiting and diarrhœa, tenderness over the stomach and spleen, and jaundice also may be present. The temperature rises rapidly to 103° to 106° and remains high for a longer time than in intermittent fever, the hot stage lasting in severe cases from 6 to 18 or 20 hours.

During this time the patient suffers from headache, pains in the back and limbs, great thirst, and gastric irritability. A remission now succeeds. The temperature falls two or three degrees, but not to

normal; free sweating occurs, the nausea and vomiting cease, and the patient becomes much more comfortable. He may fall asleep from exhaustion, but if awake is conscious of weakness, aching in the limbs, and perhaps nausea. In the course of some hours the temperature again rises, often to a higher point than before, but frequently without an antecedent chill. The same subjective symptoms are repeated, and another remission follows. Daily paroxysms usually occur, those on alternate days being severer. The temperature often reaches its highest point at the third paroxysm. The disease generally runs its course in from nine to twelve days, but it may last much longer. The type of fever may change to intermittent, which is a favorable sign, or become continued and again remittent, or remain remittent throughout; finally, the fever may subside gradually, or, less commonly, by crisis. The urine is febrile but not albuminous.

Pernicious Malarial Fever. This, as the name implies, is a form of malarial fever with destructive tendency. It is also called malignant and congestive fever. It may be intermittent or remittent. Nearly 24 per cent. of the cases occuring in the U. S. Army from May 1, 1860, to June 20, 1866, proved fatal.

Bemiss[1] divides it into three classes: (1) the algid, or congestive form; (2) the comatose form; (3) the hemorrhagic form. To this another class, (4) the gastro-enteric form, may be added. It is important to remark that the first paroxysm does not usually, in any of these forms, indicate that the type of the disease is pernicious. The first seizure may, however, prove fatal.

1. The *algid form*, according to Bemiss, occurs more frequently than any other, its perniciousness being due to an aggravation of the cold stage of an intermittent attack. The patient is extremely weak, with cold extremities, pinched features, blue lips, and faint voice. Respiration is shallow, the pulse rather slow, feeble, and irregular; the patient is further exhausted by vomiting and liquid, offensive diarrhœa, the passages sometimes being involuntary. There may be copious perspiration, but the internal temperature is very high. The mind may be clear, or deep stupor be present. Unless speedy relief can be afforded, the attack ends fatally.

2. In the *comatose form* the patient is completely unconscious, the skin hot "and of a muddy, semi-jaundiced hue" (Bemiss). Both pulse and temperature are increased in frequency. In other cases coma is preceded by wild delirium, resembling acute meningitis.

The comatose form is most apt to occur in those who continue to reside in a malarious region without proper safeguards against its poisonous influences.

3. In the *hemorrhagic form* there has been, as a rule, previous alteration of the blood, the bloodvessels, and other tissues, by long-continued malarial poisoning or cachexia. Then, when intense congestion of these parts occurs as the result of the surface chill, hemorrhage follows. In some districts, however, and at certain seasons, there has been a special predilection of the poison for the kidney, with resulting hæmaturia.

[1] Pepper's System of Medicine, vol. i. 666.

The prominent symptoms are a prolonged chill with high temperature; nausea and vomiting, sometimes of a greenish-black fluid; œdema of the lower extremities; general anasarca and occasionally œdema of the lungs, and hydrothorax; bloody and albuminous urine, with tube-casts; and intense jaundice. Pain in the right hypochondrium or over the kidneys is common.

Bemiss asserts that uncomplicated malarial fever has not a hemorrhagic tendency.

4. The *gastro-enteric form* has for its prominent symptoms nausea, vomiting, diarrhœa, intense thirst, extreme restlessness, a frequent, feeble pulse, and urgent dyspnœa. "The breathing is deep-drawn; to each expiration succeeds two short inspirations" (Da Costa). The patient is cold and partly collapsed. Reaction may or may not occur.

The patient may have several paroxysms of pernicious malarial fever, and succumb in any one of them. Convalescence is slow. The most frequent sequelæ of malarial fevers are anæmia, neuritis and paralyses, and malarial cachexia.

Malarial cachexia occurs especially in those who have lived for a long time in malarious regions. They may or may not have had typical malarial attacks. The patient suffers with dyspepsia and constipation, with occasional bilious attacks; the face is of a pale lemon-yellow color, and may be slightly jaundiced; there is marked anæmia, with pigment and crescentic and flagellate forms of plasmodia in the blood; together with great enlargement of the spleen (ague-cake) and some enlargement of the liver. The patient is weak and languid, and sometimes has considerable mental depression.

Typhoid fever is distinguished from pernicious malarial fever by its gradual onset, the absence of chills and vomiting as a rule, and on the other hand, the presence of epistaxis, delirium and ataxic symptoms, tympanites and diarrhœa with pale-yellow watery stools, and rose-colored spots. The temperature in typhoid is more continuously high, the daily oscillations being of shorter range. A history of exposure to malarial infection and of previous attacks can often be obtained. The urine of typhoid exhibits the diazo reaction, that of malarial fever does not.

Yellow Fever.

An acute specific contagious miasmatic disease, endemic and epidemic on the tropical and subtropical shores of the Atlantic Ocean, characterized by a sudden onset, a duration of a week or less, a characteristic facies, a fall in the pulse rate preceding a fall in temperature, and by albuminuria, jaundice, and vomiting, with a tendency to hemorrhages.

Yellow fever is endemic in Havana and other seaport cities of Cuba, and in Rio Janeiro, Brazil. From these centres it is liable to become epidemic, and to be carried in ships and by persons and clothing to other places. In this way epidemics have developed in the seaports of the United States, especially in the south around the Gulf of Mexico, but sometimes as far north as Philadelphia and New York. The disease becomes epidemic in the hot season and ceases upon the appearance of frost. The specific germ has not yet been isolated.

In countries in which the disease is endemic it is the custom to regard the native children as immune. Dr. John Guitéras, however, is strongly of the opinion that the disease is kept alive between epidemics by cases among these very children. He has also shown that it prevails among white children before it becomes epidemic among adults.

The period of *incubation* varies from a few hours to two weeks. Guitéras states that the cases in which it extends beyond the seventh day are exceptional.

The *invasion* is abrupt, and occurs usually in the night. It is marked by chilliness oftener than by a decided chill. The temperature rises rapidly to 102° to 103° or 104°, not often higher in favorable cases. The pulse is correspondingly increased in frequency at first, but very commonly begins to fall before the temperature, so that later the pulse is relatively slow. The face is peculiar and characteristic—it is flushed and somewhat swollen; the eyelids are somewhat swollen, with reddened edges; the eyes are watery, glistening, and slightly but distinctly tinged with yellow; the pupil is small and brilliant. Guitéras says: "The appearance of the face is often sufficiently characteristic on the first day of the disease to warrant a positive diagnosis." He also says that these phenomena are often better observed at a slight distance than on close inspection.

The *tongue* is large, moist, and coated with white fur. The stomach is irritable and the epigastrium tender. Nausea with repeated vomiting occurs. The fluid is at first of a light greenish-yellow, subsequently becoming decidedly bilious. The bowels are constipated.

The *urine* almost invariably contains albumin at some time during the first three days. Its presence may be very transient. It may be found in the evening and not at other times. The amount of albumin is sometimes very large, and abundant blood and tube-casts are found. The nephritis subsides rapidly, without leaving traces. The urine is acid in reaction and scanty in amount. It is sometimes suppressed.

During this febrile period the patient complains of headache, pains in the back and limbs, and intense thirst. The mind, however, is usually perfectly clear. Contrary to expectation, Guitéras asserts that the nervous symptoms are, perhaps, more prominent in the adult than in the child. "The loquacity, the short-cut phrases and precipitate speech, the excitement, the show of indifference with unmistakable evidences of fear—all these, that are such prominent features of the disease in the adult, are absent in the young."[2]

In from two to five days the temperature falls to or below normal, headache and pains in the limbs disappear, and the patient is cheerful and thinks himself convalescent. This is the fact in mild cases, but in more severe cases there is a return of symptoms in a few hours or at most a day or two. The jaundice deepens, vomiting becomes more urgent and in adults is accompanied by much retching. It is bilious, streaked with blood, or thick and wholly black ("black vomit"); the temperature may rise again as high or higher than in the original

[1] "Report of the Surgeon-General of the Marine-Hospital Service, 1888;" Keating's Cyclopædia of Diseases of Children, 1889, vol. i.
[2] Keating's Cyclopædia, loc. cit.

paroxysm, or it remains depressed. In any event the pulse is apt to be slow, often from 40 to 60. The urine contains albumin, blood, and casts, and may be suppressed, adding uræmia to the other toxæmia. Convulsions at this stage are usually uræmic. Hemorrhages may occur from any mucous surface. The gums are tender, swollen, and bleed easily. There may be epistaxis, hemorrhage from the ear, bowel, uterus, or vagina. Pregnant women miscarry. Ecchymoses also may form. Death may take place in coma or convulsions. If the patient linger beyond the fifth or sixth day he sinks into a typical typhoid state, with diarrhœa and marked adynamia, from which he may or may not emerge.

As in scarlet fever, the patient may be smitten down and die in a few hours from the time he was in apparent health. In other grave cases the temperature remains high, and rises instead of falls on the third or fourth day. The *duration* of the disease is from two to five or six days; if a typhoid state develop it may last ten days or two weeks.

Complications are not common. Phlebitis and lymphangitis occur, and Guitéras says he has noticed hepatitis, insanity, and paralysis (probably neuritis). Second attacks are extremely uncommon.

Diagnosis. Yellow fever is distinguished from pernicious malarial fever by the slow pulse, the characteristic facies, the early transient albuminuria, the deep jaundice, the absence of diarrhœa, the occurrence of black vomit, the tendency to hemorrhage, and the clear mind.

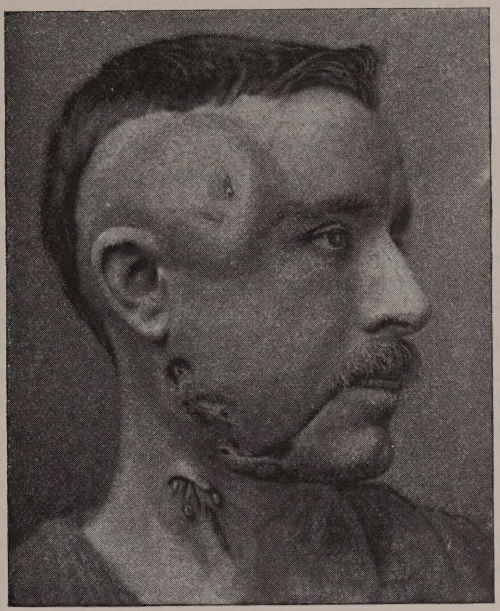
Actinomycosis.

A specific infectious disease of cattle, occurring occasionally in man, attacking especially the lower jaw, lungs, and intestine, and characterized by a long duration, by the development of tumors and metastatic growths, and by pyæmic symptoms.

It is due to the actinomyces or ray fungus (see Fig. 146), which produces in cattle the disease known as big or lumpy jaw and swelled head. The fungus is conveyed in the food or drink, and gains entrance to the body through abrasions in the mouth or a decayed tooth, or is inspired into the lungs.

At the seat of invasion a slowly growing, slightly painful tumor develops. Bones are affected as well as soft tissues. These become swollen and suppurate, the fungus being at all times obtainable. The fungous masses appear to the unaided eye as particles of yellow sand, and are greasy to the touch. When the lungs are involved the symptoms are those of purulent bronchitis or phthisis, actinomyces being found in the sputa. The masses which form upon the intestinal mucous membrane may lead to suppuration and perforation of the intestine. Metastasis to any organ may occur, with resulting local symptoms. The duration depends upon the organs involved in metastases. If these do not lead to early death, that result is brought about at the end of months or years by slow pyæmia, with resulting amyloid degeneration and its consequences. The prognosis depends upon early recognition and complete removal.

Fig. 146.



Case of actinomycosis.

Glanders.

An infectious constitutional disease, transmitted from horses to man, appearing in an acute and chronic form, and characterized by an eruption, ozæna, small tumors, ulcerations, cough, and death in coma or collapse in from one to four weeks in the acute form, or in three or four months in the chronic form, the symptoms in the latter resembling at times syphilis and at times tuberculosis.

The disease is rare in man. It may be acquired by direct inoculation of an open wound with the pus from a glanderous ulcer or nasal mucous membrane, or indirectly from infected straw or other material. The raw meat of a glandered animal also has infective power.

In *acute glanders* the onset is marked by headache, slight fever, and pains in the limbs. If a wound has been infected this becomes painful, swollen, and behaves like any poisoned wound. Sometimes a diffuse redness, resembling erysipelas, spreads from the infected point. Fagge refers to a case in which the first complaint was of pain in the side and dyspnœa, so that acute pleuro-pneumonia was suspected.

An eruption, consisting first of papules, which rapidly become flat vesicles and then pustules or bullæ, appears in the first day or two, or sometimes not for a week or even longer (Fagge). The bullæ or pustules rupture and give vent to a thin purulent discharge.

There may be hard, painful lumps in the muscles, with subsequent suppuration (farcy).

Ozæna is not always present. It appears in the second or third week of the disease. It consists of a muco-purulent, then purulent, fœtid discharge from the nose. The latter subsequently swells and becomes red and very painful. Ulcers and even necrosis of the septum are the lesions; the same catarrhal condition may exist in the throat, eye, larynx, and mouth, accompanied at times with ulcers and false membrane. The patient gradually sinks into a septicæmic condition, with irregular fever, dry brown tongue, albuminuria, delirium, coma, and collapse.

The duration of the acute form is from one to four weeks. Only one in thirty-eight cases collected by Bollinger ended in recovery.

In the *chronic form* there are ulcers upon the hand, face, forehead, or elsewhere. In other cases the lesions are abscesses in connection with joints which are followed by persistent fistulæ. In still other cases there is a pustular eruption. Ozæna may or may not exist. In still other cases the prominent symptoms are cough, bloody expectoration, hoarseness, fever, and emaciation. Bollinger reports seventeen recoveries in a total of thirty-four cases of chronic glanders.

Diagnosis. Acute glanders is distinguished from rheumatism by the history of the case, the occupation of the patient, the existence of an open, irritable sore, and the fact that while the joints may be painful, they are rarely red and swollen, as in rheumatism. Subsequently the appearance of pustules, bullæ, and ozæna makes the case clear.

The same peculiar features serve to distinguish it from pyæmia, malignant pustule, and other infectious diseases.

In chronic glanders, as suggested by the Messrs. Gamgee, an ass or horse might be inoculated with the nasal mucus or pus from a farcy.

Anthrax.

Anthrax, malignant pustule, charbon, splenic fever, etc., are names given to an acute infectious disease derived principally from herbivorous animals, and characterized by the development of a pustule or boil, with extensive brawny œdema and subsequent toxæmia; or toxæmia may appear first and metastatic abscesses subsequently. The disease also attacks the gastro-intestinal mucous membrane and the lungs.

Anthrax is caused by the anthrax bacillus and its toxins. Outside the body it forms endogenous spores, which are extremely tenacious of life, and to which infection is invariably due. They infect not only the carcasses of animals, but also the soil, all utensils used in the care of the animals or the soil, and they persist with infective power in the hides, hair, hoofs, and wool ("wool-sorter's disease"). It is possible that it may be transmitted to man by stings of insects, particularly flies and mosquitoes.

The period of incubation varies from a few hours to several days. In the form known as malignant pustule the patient has a pricking or burning feeling, which may lead him to think he has been stung by an insect at some exposed part of the body, particularly the hand, face, or

neck. At the seat of irritation first a papule, then a vesicle, develops. The vesicle may attain considerable size. The contained fluid quickly passes from clear to bloody, and then escapes, leaving a dark-brown or black scab (anthrax).

The original vesicle may be surrounded by a series of smaller ones. Instead of disappearing, the base of the vesicle becomes inflamed and indurated, the induration extending to surrounding tissue and causing a condition of brawny œdema. A whole arm or one side of the face and neck may be swollen. There may or may not be an associated lymphangitis.

The general health does not suffer at first, but in a day or two fever sets in, accompanied with delirium, sweating, great weakness, enlargement of the spleen, severe pains in the limbs, and diarrhœa. Death, preceded by collapse, may occur in from five to eight days (Fagge), or the tissue occupied by the pustule may slough out.

Bollinger and others have called attention to anthrax œdema, in which there is no pustule but only a yellowish or greenish swelling of the tissues. It is seen most frequently in the eyelids.

Anthrax of the gastro-intestinal mucous membrane, as described by Bollinger, presents the following symptoms: the patient first complains of malaise, loss of appetite, pains in the limbs, giddiness and headache. Then vomiting may set in, and a more or less severe diarrhœa, the evacuations often containing blood. There may be pain in the abdomen, which becomes somewhat tumid; the spleen is enlarged. Dyspnœa and lividity appear, with restlessness and with excitement or stupor. Epileptiform convulsions may occur, the upper limbs may be affected with tetanic spasms, there may be opisthotonos, and the pupils may be widely dilated. The pyrexia is slight, and death is preceded by extreme collapse. The duration of the disease is usually from two to seven days, but sometimes it is scarcely twenty-four hours.

Still another form of anthrax occurs among the wool-sorters of Bradford, England; it is characterized by intense dyspnœa and a feeling of oppression or constriction. Breathing is labored, but not much accelerated. Only a few coarse râles are to be heard on auscultation. The expectoration may be abundant and bloody, or absent. There is a tendency to collapse, with cold bluish skin, and a subnormal axillary temperature. The rectal temperature, however, is raised two or three degrees. Death may occur in coma and convulsions, or suddenly, the mind being clear. The duration of the disease is from one to five days. Dr. Bell says that those who survive for a week generally recover.

Bacillus Anthracis. This is found in the pus of the lesions of anthrax or malignant pustule.

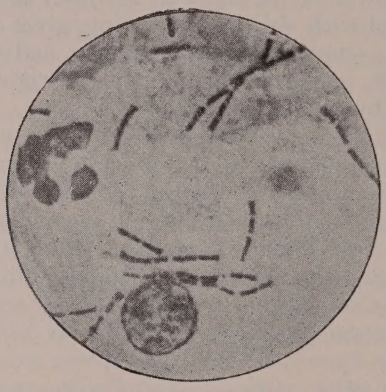
Morphology. A bacillus, 2 to 3 μ up to 20 to 25 μ in length and 1 to $1\frac{1}{4}$ μ in breadth. The bacilli are often joined end to end in long threads, and these threads are massed together in bundles. As found in animals they are short rods with square ends. They stain best with Löffler's blue, but also with the basic anilines and by Gram's method. When in the stage of spore-formation the threads look like strings of beads.

Biological Properties. It is aërobic, non-motile, and liquefies gelatin. (See Plate I., Fig. 2, A, and Fig. 147.)

It grows best in neutral or slightly alkaline media (gelatin, agar, milk, meat-infusion, etc.) at 20°–38°. The growth-limits are 12° and 45°.

Cultures on agar are quite characteristic, consisting of a dense central mass with twisting and crossing bundles all around it. In gelatin stab cultures a fine branching threadwork grows out alongside the puncture. The gelatin soon liquefies and the bacilli settle in white

FIG. 147.



Bacillus anthracis in the blood of a guinea-pig. × 1040. (GIBBES.)

masses. The growth is abundant on potato, and is grayish, dry, rough, and irregular. The virulence is attenuated by cultivation. Drying does not kill the spores. Very toxic substances are found in the culture medium. When *inoculated*, the organism produces the pustule of anthrax.

Anthrax bacilli are not so numerous in human blood as in that of the lower animals. They are most likely to be found in the spleen, which is apt to be much swollen.

DIAGNOSIS. In doubtful cases a mouse or guinea-pig should be inoculated with the blood. *Carbuncle* is distinguished by its tendency to develop upon the back or shoulders, and other covered portions; anthrax on uncovered portions. In carbuncle there is a series of openings resembling a sieve, filled with pus and plugs of necrotic tissue. In anthrax there is at first a central black crust. The boggy feeling of carbuncle is different from that of the brawny œdema of anthrax. Finally, in carbuncle, anthrax bacilli are not found in the blood.

The intestinal and thoracic forms are distinguished by the occupation of the patients, the absence of other adequate cause, and the result of the blood examination.

Foot-and-Mouth Disease.

A specific infectious disease, communicated to man through cattle, sheep, or pigs, and characterized by a stomatitis. It is communicable

by milk; the period of incubation is from three to five days. Invasion is characterized by slight fever, heat and soreness of the mouth, and the development of vesicles which burst and leave shallow ulcers. Saliva is freely poured out. The tongue swells greatly, and eating is painful. Vesicles sometimes appear about the fingers, but not upon the feet. The disease lasts from one to two weeks, and ends almost invariably in recovery.

Hydrophobia.

An acute specific disease communicated to human beings by the bites of animals similarly affected. The animals most frequently affected are the dog, fox, wolf, cat, and skunk; 90 per cent. of the cases in human beings are due to dog-bites.

The period of *incubation* is uncommonly long and very variable—from two weeks to two months usually. It is said in some cases to be a year or more. The disease has been divided into three stages—the melancholic, the spasmodic, and the paralytic.

In the *melancholic stage* there is pain, hyperæsthesia, or even reopening of the healed wound. The patient is extremely depressed in spirits and may be irritable. He seems to be laboring under a constant tension of fear and keenly sensitive to light, sounds, or draughts. He is affected with thirst, but attempts to swallow water cause intensely painful spasm of the larynx.

The *second stage* is reached usually on the second day. The laryngeal spasms are increased and lead to intense dyspnœa and to pitiable struggling and gasping on the part of the patient. In addition to the convulsive seizures, the patient foams and froths at the mouth, and his face expresses the extreme terror and mental anguish he feels. The second stage lasts from one to three days, and is followed by exhaustion intermitting with paroxysms of less severity. The patient may now be able to swallow easily, but there is great weakness of the heart, and death may occur from failure of the heart, from asphyxia, or in a convulsion. The duration, as indicated, is only a few days. The result practically is always fatal, but recovery may be possible. Bites of the face are the most likely to be fatal.

The Plague.

An acute specific infectious and contagious disease, occurring in epidemics, characterized by high fever, sometimes by petechiæ and other hemorrhages, and in cases which last long enough, by buboes. The death-rate is extremely high.

The plague is a disease of the East, being endemic in some parts of India, but epidemics have occurred in Italy, Russia, Turkey, England, and other parts of Europe.

The period of *incubation* is from two to seven days. The invasion is marked by lassitude, languor, headache, and dizziness. The stupid aspect and staggering gait may lead to the belief that the patient is drunk. Chill or chilliness soon supervenes, followed by fever, which often rises to hyperpyrexia, and is accompanied with unquenchable thirst,

and sometimes nausea and vomiting. Delirium and a typhoid condition follow, with a marked tendency to failure of the circulation and collapse. If the patient survive until the second or third day, glandular swellings develop in the groin, or axilla, or angle of the jaw. Often they have to be sought for to be found. Sometimes they are prominent and are followed by suppuration and even ulceration. Carbuncles are much rarer manifestations than buboes. Petechiæ, vibices, hemorrhages into the kidney, bloody vomit, occur in the worst cases.

The duration is from six to ten days. If there is much suppuration convalescence is prolonged.

Leprosy.

A chronic specific infectious disease, characterized by the development of tubercles, anæsthetic patches, and neuritis, and followed by ulceration and destruction of tissue. The disease occurs especially from puberty to the thirtieth year, and oftener in men than in women. It develops slowly and insidiously. Sometimes the first skin lesion is a crop of bullæ, suggestive of pemphigus. More commonly there appear reddish or violet-colored patches, varying in size from a quarter of an inch to two or three inches in diameter, and becoming of darker hue later. The next step is the formation of nodules, which are characteristic of the disease. These may develop upon the patches already described, or in other places. They vary in size from a pea to a bird's egg or larger. They are most common upon the face and extensor surfaces of the arms, legs, fingers, and toes. The tubercles consist of an infiltration into the true skin; they are raised, firm, relatively painless, and vary in color from red to copper. The face is characteristically distorted into a fierce expression (leontiasis). The tubercles may become absorbed and leave atrophic areas, but generally they break down into eroding ulcers, which slowly burrow and increase in extent, eating off portions of the nose, fingers, hands, and feet, and exposing muscles, tendons, nerves, bloodvessels, and bone. Tubercles form also upon nerve trunks and ulcers upon mucous membranes.

In other cases or in combination with the tubercles are anæsthetic areas, especially upon the limbs and trunk. Ulcers may follow without the previous occurrence of tubercles. With the anæsthetic patches are associated crops of bullæ and neuritis.

The further peculiarities of the disease are its long duration; its slow progress interrupted by apparent recovery of some of the ulcers; its afebrile course (the temperature is generally subnormal); its comparative painlessness, and its slight impairment of the general health. Death results from gradual wasting, or is hastened by some intercurrent affection.

The specific cause of the disease is probably the bacillus lepræ of Hansen. It is found in the thin pus of the ulcers and in the lesions themselves. It consists of rods 4 to 6 μ long and 1 μ broad, closely resembling tubercle bacilli. They may be distinguished by their yielding their color more readily, and taking easily aniline dyes in simple watery solution (Von Jaksch). (See Plate I., Fig. 4, B.)

The *diagnosis* from a tubercular *syphilide* is made by the history of the case, the possibility of infection, the bacteriological examination, the slow progress, and the inadequacy of specific treatment. The presence of anæsthesia and of neuritis points to leprosy.

Miliary Fever.

Miliary fever, or sweating sickness, is an infectious disease, occurring in epidemics, and characterized by moderate fever, profuse sweating, tenderness and a sense of oppression at the epigastrium, and a vesicular eruption. The disease has occurred epidemically in England, but is not met with now outside of France and Italy.

After mild prodromal symptoms the disease sets in suddenly with moderate fever, profuse sweating, and epigastric distress, sometimes amounting to anguish. The characteristic eruption appears on the third or fourth day. It consists first of small reddish maculæ, in the centre of which a vesicle develops. The latter varies in size from a pin-head to a pea. The contents are at first clear, but subsequently become purulent. Desiccation and desquamation follow. The eruption is most profuse generally upon the neck and trunk. Sometimes there are marked nervous symptoms and even convulsions and fatal collapse.

It is distinguished from rheumatism by the moderate fever and absence of joint swellings, and from malarial fever by the absence of chills, of periodicity in the febrile movement, and absence of malarial organisms from the blood.

The duration of the disease is from one to four weeks. The mortality in some epidemics has been very high, in others very low.

Milk Sickness.

An acute disease affecting cattle, and transmitted from them to human beings in the milk or meat. The disease is limited to a few sparsely settled localities west of the Alleghany Mountains. It is characterized by great debility, with muscular tremor upon motion (hence the name "trembles"), vomiting (hence "puking fever"), a peculiar fœtor of the breath, obstinate constipation, and moderate fever or subnormal temperature. The vomited matters are said to be of a peculiar soapy material of yellowish or greenish color. The duration is usually less than a week. The patient may sink into a typhoid condition and die in coma, or he may die in a few hours. Convalescence is protracted.

Trichinosis.

An acute infectious disease, caused by absorption of trichinæ spiralis, and characterized by gastric and intestinal irritation, followed by pain and stiffness in voluntary muscles, œdema of the eyelids, face, and feet, by profuse sweating, and by death or tardy convalescence.

The trichinæ are absorbed by human beings through raw or imperfectly cooked food, often in the form of sausage. The trichinæ are en-

cysted when absorbed, but within forty-eight hours they are liberated in the intestine and can be found adherent to the mucous membrane. In the course of six or seven days, each liberated female worm produces about 180 embryos, which immediately penetrate the walls of the intestine and travel or are carried to all parts of the body, becoming in turn encysted.

Swallowing of trichinous flesh does not necessarily produce symptoms; the trichinæ may be destroyed in the stomach, or, if calcified, may pass through the intestine unchanged. When symptoms result, they depend upon the number of trichinæ which become liberated. The symptoms produced are sleeplessness, lassitude, anorexia, nausea, vomiting, tenderness over the abdomen, and diarrhœa. The symptoms may be so severe as to cause death in two or three days. If the patient survive, toward the end of the week the voluntary muscles become stiff, painful, and contracted. The muscles feel hard and swollen. The eyelids, face, and sometimes the feet become œdematous. Depending upon the muscles involved, there are interferences with the eye movements, contractions of the jaw muscles, difficulty in breathing or in swallowing, etc. The calves of the legs are especially involved. Recurrent œdema over the affected muscles, eyelids, and face, is very common and characteristic. Profuse sweating also is very common, and at times there are severe neuralgic pains.

The fever is usually moderate, but it may be high. The pulse is very frequent if trichinæ reach the heart. The later stages in fatal cases are marked by insomnia, delirium, stupor, and coma.

The duration varies from a few days to four or five weeks or even longer. Muscular pains may persist for months after recovery. Death results from exhaustion, or from some complication, as pneumonia or ulceration of the large intestine.

Diagnosis. It is distinguished from *typhoid fever* by the presence of vomiting and œdema of the face and eyelids, the development of muscular troubles, by the absence of hebetude, delirium, and other typhoid symptoms, and of the characteristic eruption and enlargement of the spleen.

Muscular rheumatism is distinguished by being limited to one part, as the lumbar region, arm, or chest; by its appearance following exposure to a draught; and by the fact that it is not preceded by nausea, vomiting, and diarrhœa, nor accompanied with œdema.

Constitutional Syphilis.

Constitutional syphilis may be acquired or congenital.

Acquired syphilis is characterized, first, by the initial lesion, or chancre, which appears usually in a week after contagion; second, by a period of incubation generally lasting six weeks, but varying from one to three months; third, by so-called secondary symptoms, comprising febrile symptoms, polymorphous skin eruptions, ulcers upon the tonsils, adenitis, less frequently mucous patches in the mouth, or condylomata about the anus, iritis and retinitis, and loss of hair. The lesions of this period are symmetrical. Fourth, after an interval varying from several months to twenty years, by so-called tertiary phenomena,

which manifest themselves in some cases. These are due to chronic inflammatory indurations of the skin and subcutaneous tissue, resulting in suppuration and ulceration; or of the bones, producing periostitis and necrosis; or of organs, producing gummata and cirrhosis; or of the nervous system, resulting in gummata or chronic degenerative changes. The lesions of this period are unsymmetrical.[1]

The course of syphilis in different persons varies as widely as with any of the eruptive fevers. In some the chancre is a mere papule which heals almost unnoticed; no secondary symptoms appear, and tertiary symptoms also are altogether wanting, or a chronic degeneration of the nervous system develops after the lapse of many years, the patient in the meantime remaining in apparent health. All this may occur, too, without the aid of specific treatment. In other cases the disease is malignant; tertiary symptoms appear very early or appear to take the place of secondary symptoms; ulceration may rapidly melt down and destroy the alæ of the nose or the soft palate; or rebellious periostitis with necrosis may attack the tibiæ, the nasal bones, or the cranium.

In an ordinary case of acquired syphilis, in about six weeks after the appearance of the chancre the patient complains of languor, weariness, slight fever, pains in the bones, impaired digestion, and a tendency to anæmia. An eruption now appears. It is most marked on the trunk and upper extremities, especially the chest and forehead (corona Veneris). The eruption may be roseolous, squamous, vesico-papular, papular, pustular, bullous, or tubercular. The color has been aptly compared to that of a slice of raw ham. The enlargement of the inguinal, epitrochlear, and post-cervical glands, which precedes the eruption, persists. Shallow ulcers with a sharply-defined grayish outline appear on both tonsils. They are painless and do not spread. Ulcers are also liable to appear upon the pharynx, buccal surfaces, tongue, angles of the mouth, penis, vulva, vagina, and around the anus. In the mouth these are apt to be very painful, and may persist in spite of treatment for weeks or months. Relapses are not uncommon. Sometimes there are raised white patches upon the pharynx. Sometimes the hair becomes very thin and falls out, leaving the patient without eyebrows and more or less bald. Iritis and retinitis are usually later symptoms. Other symptoms occasionally occurring at this stage are periostitis, usually slight, and onychia.

The most common of the symptoms enumerated are the eruption and the tonsillar ulceration.

The eruption comes out gradually during two or three weeks and persists for about two months. Rarely, however, it is fleeting, or, on the other hand, is unduly prolonged.

The secondary symptoms last from six to eighteen months. After their disappearance the patient may remain entirely well for life. In other cases after apparent health, lasting for months or years, the tertiary phenomena already mentioned appear. In the interval the patient may have suffered with various local skin eruptions or with ulcers upon the buccal mucous membrane.

[1] *Fever* is a constant accompaniment of all forms of syphilis. (See Fever.)

For a description of the tertiary lesions of syphilis see works upon surgery, and other articles in this book upon visceral diseases in the causation of which syphilis is a factor.

Hereditary syphilis differs in some respects from the acquired form. At birth the syphilitic infant usually exhibits no evidence of its inherited taint. In the course of from one to twelve weeks it develops a catarrhal inflammation of the nasal mucous membrane, which causes snuffling in breathing, and hence is called "snuffles." An eruption soon appears, symmetrical in distribution. It is most frequently erythematous or papular, but it may be squamous, vesicular, pustular, or bullous. It is more apt to be moist and to favor the genitalia and flexures of the thigh than in acquired syphilis. It is of the same ham-color as in acquired syphilis. Coincident with the "snuffles" and eruption appear stomatitis and ulcers at the angles of the mouth, and sometimes condylomata around the anus. Meantime the child has begun to waste, to be peevish, to be anæmic, and gradually to assume the appearance of a wizened, dried-up old man. As in acquired syphilis, there may be iritis, though it is uncommon, and inflammation of the other structures of the eye, but nodes and disease of the liver are rare. The infant very frequently dies during this period from exhaustion and inanition.

If the child survive for a year, the secondary symptoms usually disappear and the disease becomes latent. Relapses may occur, and in them, according to Mr. Hutchinson, condylomata are liable to appear. The same observer states that the tertiary period may begin at any time after the fifth year, but it is commonly delayed till about the period of puberty. In the meantime the patient may appear fairly well, but usually his development is retarded, there is a tendency to anæmia, and he has often naso-pharyngeal catarrh, flattening of the bridge of the nose, premature decay of the upper incisor teeth, and protuberant forehead.

The teeth may be perfectly normal, in other cases characteristically syphilitic. The malformation affects especially the upper central incisors of the permanent set. It was first described by Mr. Hutchinson. It "consists in a dwarfing of the tooth, which is usually both narrow and short, and in the atrophy of its middle lobe. This atrophy leaves a single broad notch (vertical) in the edge of the tooth, and sometimes from this notch a shallow furrow passes upward in both anterior and posterior surfaces nearly to the gum. This notching is usually symmetrical. It may vary much in degree in different cases; sometimes the teeth diverge, and at others they slant toward each other." (See Fig. 75.)

Further, the patient may have had or may now be attacked with keratitis, affecting both eyes, producing cloudy opacities and being accompanied by great photophobia. Again, there may be nodes upon the long bones, with nocturnal exacerbations of pain. Cerebral deafness, according to Hutchinson, is not rare, but cerebral blindness is. There may be ulceration upon the legs, and periostitis and necrosis. The patient usually recovers completely, but he is more liable to be carried off by intercurrent disease than a healthy person, and in general has less resisting power, especially to tuberculosis.

Tuberculosis.

Tuberculosis is an infectious disease, the course of which may be acute or chronic. It is caused by the bacillus tuberculosis. This micro-organism sets up a specific inflammation characterized by the development of nodules or tubercles, or by a diffuse growth of tuberculous tissue. Either anatomical product may undergo caseation or sclerosis, and in either instance, ulceration or calcareous degeneration.

Invasion of the body by the micro-organism may give rise to general infection, with an eruption of miliary tubercles in most of the organs and structures of the body, or to a local infection. General tuberculosis is acute; local tuberculosis may be acute or chronic. In acute tuberculosis the serous membranes, the lungs, liver, kidneys, lymphatic glands and spleen, the bone marrow and the choroid coat of the eye may be invaded in whole or in part. In chronic tuberculosis the lymph glands, the lungs, the serous membranes, the tissues and organs of the alimentary canal, the liver, the organs of the genito-urinary system, and the brain and cord are individually invaded.

The diagnosis of any form of tuberculosis is aided by the determination of the chief factors in its etiology, where this is possible. First. The discovery of the bacillus tuberculosis in any inflammatory area, or any product of inflammation, as serum, blood, pus, or the secretion from any gland or mucous membrane invaded by the disease, establishes at once the diagnosis of this condition. The method of determining the presence of this micro-organism is fully detailed in the various descriptions of tuberculosis in the discussion of local diseases, and in the accounts of the examination of the sputum and of exudations and transudations. Second. As tuberculosis is an infectious disease, discovery of the infection is an aid in the diagnosis. Infection takes place by means of the inhalation of the sputum or other secretions, which when dry float about in the air. It implies in a measure more or less contact with individuals previously infected. In rare cases such contact is productive of the disease by means of direct contagion. The second source of infection is the food supply. This may occur from the consumption of milk secured from a cow infected with tuberculosis. The eating of meat of tuberculous animals may possibly lead to infection. Direct inoculation is another but rarer source of infection. This usually occurs accidentally only. Finally, it is possible that tuberculosis may be inherited. A more prominent ætiological factor, which aids in the diagnosis of the disease is the presence of a certain type of structure which is a marked hereditary characteristic in families, on account of which feeble resistance is offered to the invasion of the tubercle bacillus. The phthisical or phthisinoid chest which belongs to this type has been described elsewhere, and the tuberculous and scrofulous states outlined (see pages 55 and 234). These anatomical conditions, which are inherited, undoubtedly favor the development of tuberculosis.

It is a mistake to lay much stress in the diagnosis of tuberculosis upon the age or the occupation of the individual. Tuberculosis may occur at any age. It is true, however, that at certain periods of life the tubercles are distributed more commonly in one group of organs,

while in other periods it affects another group. Lymphatic, joint, and meningeal tuberculosis is most common in the first decade of life. The mesenteric glands are particularly open to invasion at this period.

The *diagnosis* of tuberculosis, whether local or general, is further aided by a complete knowledge of the phenomena that attend the entrance of the virus into the body and the mode of diffusion throughout the body after infection has taken place. The phenomena at the point of entrance of the micro-organism are nearly always distinct. The general invasion is associated with symptoms like those of specific fevers. The local secondary effects upon the tissues are always decided. It must be borne in mind that after the exposure, which may lead to infection, either an acute form of tuberculosis of a general character may be set up, with or without marked local symptoms, or acute local tuberculosis may arise. In local tuberculosis the disease is confined to one organ or to the lymphatic glands and the organs in the lymphatic distribution, as the bronchial glands, which are primarily affected, and the lungs. In these structures the entire process of nodular formation, caseation or sclerosis, ulceration or calcification, may take place. The disease remains primarily local. On the other hand, it may spread by continuity of structure through the lymphatics throughout the remainder of the organ affected, leading to its ultimate destruction and the death of the patient; or general infection of the system may take place from the primary local area. The primary seat of infection may be the lungs, the larynx, or the alimentary tract of the genito-urinary organs. Primary tuberculosis of the serous membranes, of the lymph glands, of the bones and joints, may take place.

The symptomatology and diagnosis of the various forms of tuberculosis are detailed in the section devoted to the special diseases of the various organs of the body.

Acute miliary tuberculosis has been spoken of elsewhere (see page 317). Its course may resemble typhoid fever, septicæmia, or malignant endocarditis. It usually develops in the course of tuberculosis in some other organ of the body. The typhoid form has been described in the section indicated. It must not be forgotten that the diagnosis is rendered positive by the demonstration of the presence of tubercle bacilli in the blood, or of the occurrence of choroidal tubercles in the eye-ground. Another form is attended by marked pulmonary symptoms. This is the type seen in the bronchial pneumonia that occurs in children following measles and whooping-cough (see Catarrhal Pneumonia). Of the pulmonary symptoms dyspnœa is the most prominent. Cyanosis is marked. The physical signs are not prominent, and may be those of bronchitis alone. Although there is impaired resonance at the base of the lungs, areas of hyper-resonance are observed above and in front of the chest. Collapse of the lung may cause tubular breathing. The temperature rises to 102° to 103°. An inverse type may be seen.

The diagnosis of acute tuberculosis is determined by the history of infection from extraneous sources or from local tuberculosis in some portion of the body, and the presence of bacilli.

The following conditions should point to chronic tuberculosis in some portion of the body: 1, emaciation, not otherwise explained; 2,

anæmia; 3, weakness without cause; 4, fever—the temperature should be taken every two hours night and day; 5, causeless sweats; 6, gastro-intestinal catarrh; 7, morning nausea; 8, signs of local inflammation in some organ of the body.

Remarks on the Diagnosis of Infectious Diseases.

A satisfactory diagnosis is only made when a correct appreciation of the evolution of the disease and facts concerning its activity are ascertained. The eruptive fevers, particularly, are differentiated with difficulty unless the chronological sequence of the phenomena of the development of the disease are weighed. These facts, in a suspected case, relate to the history of exposure of the patient, the presence of an epidemic, the presence of conditions favorable to the development of the disease, etc. The following should be ascertained:

1. The occurrence of an *epidemic*. In a suspected case it may be of weight in aiding in the distinction of the disease.

2. The history of *exposure* to the suspected infection, either mediate or immediate, remotely in time or place, must have great value.

3. *Immunity*, acquired or artificial, to a particular disease, may exclude that affection. The eruptive fevers rarely occur a second time. One attack of erysipelas, however, predisposes to subsequent attacks.

4. *Ætiological facts* pertaining to the suspected disease, as hygienic surroundings, a malarial region, etc. Other factors bearing on ætiology, as the season in cases of typhoid fever, are often valuable in pointing to the affection.

5. *The Age*. The eruptive fevers are peculiar to childhood, typhoid fever to early adult life.

6. The *occupation* in affections transmitted from animals to man.

7. The duration of incubation, the mode of onset, the mode of development, the characters of specific features, and the day of the development of each, are important data to aid the diagnosis.

Simple Continued Fever.

A non-contagious fever, lasting from one to twelve days, not depending upon any known specific cause and not attended with definite local lesions. Its chief characteristic is the continued elevation of temperature.

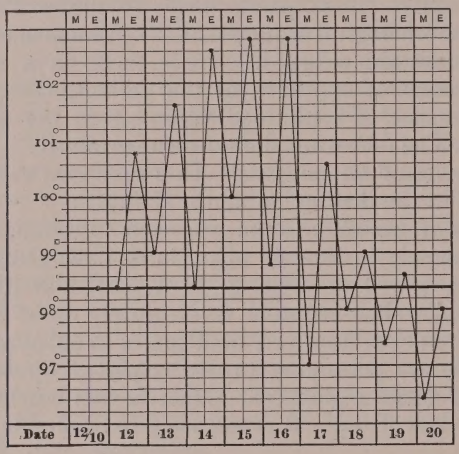
It occurs especially in children and in those prone to a ready disturbance of the heat-regulating apparatus. Great mental and physical exhaustion, prolonged bathing in the hot sun, and disturbances in dgestion may cause it. Perhaps, as suggested by Guitéras, some of the cases occurring in the tropics and in very hot weather should be regarded as very mild forms of thermic fever.

The onset of the disease is abrupt. There may be a chill, or in nervous children a convulsion; but these are rare. The temperature rises rapidly to 102°–104°, accompanied with headache, thirst, restlessness or drowsiness, loss of appetite, a coated tongue, constipation, and occasionally nausea. The urine is scanty and sometimes there is a heavy deposit of urates. There may also be more or less muscular

soreness. Sometimes within twenty-four or forty-eight hours there is free perspiration and a rapid subsidence of the fever and all its symptoms. This is ephemeral fever.

In other cases the fever continues for a week or ten days longer. During this time the symptoms already noted continue. Sleep is disturbed and mild delirium is at times present. Respiration and pulse are not much accelerated. Sudamina upon the abdomen and

FIG. 148.



Simple continued fever.

herpes upon the lips are common. Pale-bluish maculæ are sometimes seen. The spleen is not enlarged except in very rare cases, and there are no local evidences of disease. The fever subsides more gradually than in ephemeral fever, the defervescence being marked at times by perspiration, a few loose stools, a copious deposit of urates in the urine, or by hemorrhages from the nose, rectum, uterus, or urethra.

The *diagnosis* from other fevers and febrile affections is made by the absence of any characteristic eruption, of enlargement of the spleen and liver, and of any lesion, such as endocarditis, bronchitis, or pneumonia.

CHAPTER XI.

DISEASES OF THE NERVOUS SYSTEM.

Disturbances of sensibility, disturbances of motility, ataxia, disturbances of the reflexes, vasomotor and trophic disturbances, and disturbances of intellection are produced by diseases of the nervous system.

The Disturbances of Sensibility.

In *anæsthesia* there is diminution or absence of sensibility. In *hyperæsthesia* the sensibility is abnormally increased, so that even weak irritations may produce painful sensations. *Paræsthesia* are abnormal sensations in the skin, as formication, numbness, pricking, etc., which are also called symptoms of sensory irritation. Actual pain may also be one of the symptoms of sensory irritation. Such abnormal sensations are due to morbid states of the nerve itself.

Cutaneous sensibility is of several varieties, and hence to determine any change the various forms of sensation must be tested. In nervous diseases one variety of sensation may be destroyed while other sensations remain intact. Such abnormal changes are known as partial anæsthesiæ or paralyses of sensation. The following varieties of cutaneous sensibility are investigated:

1. *Tactile Sensibility.* Tactile sensibility is tested by touching the skin with the finger or a blunt object. The patient's eyes being closed, he is asked whether he has perceived the touch or not. It is well sometimes to control the experiment by asking the question without making contact with the skin. Comparative test should be made of the opposite side, which is presumably healthy. By the tactile sense the form of objects and the external characters are also judged. Smooth, rough, hard, soft, round, or angular objects are employed. The eyes must be kept shut. Familiar objects may be used; when placed in the hand the patient is made to name them if possible. Coins, keys, or wooden geometrical objects are used for this purpose.

2. *Sense of Locality.* When any part of the surface of the body is touched we can, under normal conditions, tell the exact locality of the point of contact. This ability to localize the sensation is lost by patients with nervous diseases. In addition to designating directly, or by means of the hand, the part of the body that is touched, the tactile sense is also tested by means of compasses. By this method the patient is subjected to two simultaneous irritations of the skin at the same time. The points of the compass may be distinguished as separate irritants at from 11 to 15 mm. on the cheeks, 6 mm. at the tip of the nose, 1.2 mm. at the tip of the tongue, etc. At the tips of the fingers the two points can be detected at from 2 to 3 mm.; on the thigh, 77 mm. Various

modifications of this test must be employed to control the results, as by bringing down one point at a time, or at a different place each time.

3. *Sense of Pressure.* This sense is tested by placing the hand on a firm, hard surface like a table, and placing graduated weights upon it. Change in this sense may be confirmed by employing pressure with the hand or a pencil upon the skin, using various degrees of force.

4. *Sense of Temperature.* Thermic sensibility is tested by the application of hot and cooler bodies alternately. The sensations to heat and to cold are due to distinct functions, and therefore may be separately modified. The heat sense may be abnormal, while the cold sense is often unchanged. The application of either hot or cold objects, if the sense of temperature is impaired, may not be perceived at all. The sense may be blunted, so that hot water feels as if it was simply tepid, or it may be lost entirely, the patient perceiving the touch, but not the temperature of the object applied. In partial anæsthesia to cold the application of a bit of ice may be described as causing a warm sensation. Differences of temperature are recognized by these functions. In health a difference of one degree Fahr. in the temperature of the body is usually distinguished without difficulty. The face and fingers are even more sensitive. The difference in temperature may be determined by applying vials filled with water at varying temperatures.

5. *Sensation of Pain.* Loss of sense of touch does not necessarily imply loss of sensation of pain. The former may be lost, while pain is readily excited in the affected area. The loss of sensibility to pain is known as *analgesia.* It is of common occurrence in peripheral and central nervous diseases. The point of a pin, thermal irritants, electrical currents, or pinching of the skin, are methods used to determine the sensation of pain.

6. *Electro-cutaneous Sensibility.* This is determined by faradization, but does not give any better information than is secured by testing the tactile sense and the pain sense.

7. *Delayed Conduction of Sensation.* After the irritant is directly applied in certain diseases the patient does not respond for a considerable interval of time. This delay of conduction is seen in locomotor ataxia particularly. The sensation of touch may be perceived several seconds before the sensation of pain.

8. *The Muscular Sense.* By the muscular sense we are enabled without the sense of sight to tell the position of our limbs. After any passive movements made by an observer a healthy person can tell at once the direction and character of the movements. In patients with nervous diseases this faculty may be lost. When a patient is called upon to make a definite movement, the eyes being closed, this movement is not completed, or is incorrectly made, if the muscular sense be lost.

Anœsthesia of the Skin. Any break in the conducting path from the surface of the body to the centres of sensation in the cerebral cortex causes anæsthesia, which may be complete or partial. Anæsthesia may be peripheral, spinal, or cerebral. In *peripheral anœsthesia* the terminations of the sensory cutaneous nerves do not respond to irritations. This is seen after the application of anæsthetics to the skin, or of corrosive substances, as acids or alkalies, carbolic acid, etc., or from the

use of cocaine or morphia. Another form of peripheral anæsthesia is due to disease of the nerve trunks from trauma, from compression of the nerve, or from neuritis.

Spinal Anæsthesia is seen in disease of the spinal cord, particularly when the posterior roots, the posterior columns, and the posterior cornua are diseased, as in locomotor ataxia. It is also seen in acute and chronic inflammation of the cord, and in compression or in new growths. The anæsthesia is bilateral.

Cerebral Anæsthesia is seen in hemorrhages, local softening and tumors, which affect the posterior portion of the internal capsule. When half the body is affected it is known as hemianæsthesia, and is on the opposite side of the lesion. In hysteria anæsthesia is often seen.

Neuralgia.

Neuralgia is characterized by pain in the course or distribution of the affected nerve. The pain is of pronounced severity, and occurs in remissions and intermissions. The *symptoms* of a neuralgic paroxysm may be preceded by hyperæsthesia over the part subsequently affected. The pain is of a burning or shooting character. It is usually limited to the distribution of the affected nerve, or may extend into other regions. It may be excited by external irritants, by mental excitement, and often by movement of the part. On examination, the territory of the affected nerve may be anæsthetic. Usually, however, there is hyperæsthesia of the skin. Wherever the affected nerve is accessible to pressure pain can be elicited. The nerve-trunk may be tender during the attack, and during the intervals between the attacks. Often in neuralgia there is some spasm of the muscles supplied by the nerve.

Vasomotor symptoms are common. The skin may be pale, or the area reddened. When the trigeminal nerve is affected the skin and conjunctivæ are both reddened. The secretions, as the tears, may be modified. Eruptions like urticaria or herpes may develop along the course of the nerves. Prolonged neuralgia may cause marked nutritive disturbances.

General Conditions. The patient who is the subject of neuralgia may be in apparent good health. The neuralgia may be due to constitutional causes, as rheumatism or gout; to some form of toxæmia, as malaria; to some condition of the blood, as anæmia; and may be due to trauma or cold.

The following individual forms of neuralgia are seen: 1. Neuralgia of the trigeminus, or *tic douloureux*. The fifth pair in its entirety or some of its branches are affected. The pain may be associated with twitchings; with vasomotor disturbances with eruptions, and with changes in the secretions. Trophic changes, as the hair turning gray, or atrophies, may follow. The first branch (ophthalmic); the second branch (supra-maxillary); the third branch (infra-maxillary), are most frequently affected. Points of pressure are usually readily detected at the foramina for the exit of the nerves. 2. Occipital neuralgia. 3. Neuralgia of the brachial plexus. 4. Intercostal neuralgia. 5. Neuralgia of the lumbar plexus, of which we have lumbo-abdominal, crural

and obturator neuralgia. This form of neuralgia (lumbar plexus) must not be confounded with bone and joint disease; with lumbago; renal colic; appendicitis, and uterine affections. 6. Sciatica. 7. Genital and rectal neuralgia.

Trigeminal neuralgia must be distinguished from headache due to other causes, affections of the bones and periosteum, and affections of the teeth. The distribution of the pain; the points of pressure; the paroxysmal character of the pain, aid in the diagnosis.

Disturbances of Motility.

Paralysis is a loss of power of the muscles of the body controlled by the will. It must be distinguished from loss of motion or inhibition of function due to disease of the muscle, or to pain which is excited by movement. The presence of tenderness and of pain on passive motion serves to distinguish this form of paralysis.

When there is absolute loss of power the paralysis is *complete*; when there is weakness of the muscles, it is known as *paresis*. In this latter condition certain movements are possible.

Causes. Disease in any portion of the cortico-muscular conduction-path or pyramidal tract may lead to paralysis. Destruction of the function of the motor centres in the cerebral cortex may lead to paralysis. Paralysis is also due to disease of the muscles. It is known as *myopathic paralysis.*

Paralysis of one lateral half of the body is known as *hemiplegia.* One-half of the face, the arm and the leg, or an arm and a leg of one side alone are paralyzed. The trunkal muscles are not affected in hemiplegia. Hemiplegia is invariably of brain origin. Paralysis of the lower transverse half of the body is known as *paraplegia.* It is of spinal origin. A *monoplegia* may be facial, brachial, or crural, according to the situation of the paralysis. Monoplegias are due to diseases of the brain, of the spinal cord, or of the nerve trunk. Monoplegia of cerebral origin is always cortical. Monoplegia of spinal origin is seated in the ganglion cells of the anterior cornua. A *local paralysis* is loss of power of a single muscle or group of muscles. When many local palsies exist it is known as multiple paralysis. A local paralysis is frequently due to disease of the nerve trunk—a *neuritis.*

The *symptoms* of paralysis are recognized by the patients' statements and by physical examination. 1. There is loss of power of the muscles. 2. Change in the character of the muscles. 3. Changes in the reflexes, the nutrition, and the sensations. Changes in the condition of the paralyzed muscle are valuable diagnostic criteria as to the cause of the paralysis (see page 135). 1. The paralyzed muscle may retain its normal volume and normal nutrition. 2. The muscle may be atrophied. To this class belong the *atrophic paralyses.* In the former instance the break in the ganglion of the motor fibres exists somewhere between the cortex and the cells in the anterior cornua. In atrophic paralyses the cause is seated in the ganglion cells or in the peripheral nerves. The ganglion cells must influence the nutrition of muscles. If they are normal and the nerves not affected the nutrition of the muscles remains good.

In addition to the atrophy of the muscles, the nerves proceeding from the point of lesion to the muscle atrophies or degenerates. On account of this degeneration certain reactions are brought out by electricity (see Electrical Diagnosis).

When passive motion is performed in some forms of paralysis there is resistance to the movements on account of contraction of the muscles. They are known as *spastic paralyses.* When muscular resistance is lessened they are known as *flaccid paralyses.* In long-continued paralysis *contracture* of the muscles takes place. It must not be confounded with spastic paralyses.

In paralysis of the face the mouth is drawn toward the sound side, unless contractures take place in the paralyzed muscles. In paralysis of half the tongue when it is protruded the tip turns toward the paralyzed side.

Motor Irritation. Motor irritation is indicated by spasm, which is a morbid movement excited independently of the will. Spasm is due to irritation somewhere in the motor tracts. The irritation may act directly on the nerves or be produced by an irritation in the periphery, as in reflex spasms.

Spasms may be *clonic* or *tonic.* When the muscular contraction lasts but a short time, and is followed by relaxation, the two alternating rapidly, they are clonic in character. There is constant convulsive movement. In tonic spasm there is persistent contraction of the muscle. Tonic and clonic spasms may alternate in the same individual, or the same group of muscles.

Spasms are also divided into many forms, depending upon the degree and character of the motor irritation. They are all grouped under the head of motor irritations. 1. *Epileptiform convulsions* are clonic spasms, or tonic-clonic. They may extend over the whole body, or be limited to one-half the body, or to one portion, as the arm or leg. The true type is seen in epilepsy, in hysteria, and in organic disease of the brain. 2. *Rhythmical contractions.* There is more or less continuous moderate contraction of groups of muscles. They are seen in apoplexy, in cerebral sclerosis. Such contractions occur before or after an epileptic fit. 3. *Tremor.* The spasms are moderate, rapidly succeeding one another, small in extent: when most severe, known as "shaking." Tremor is seen in paralysis agitans in its most pronounced form. We also have senile tremor, alcoholic tremor, hysterical tremor, and tremor due to metallic poisonings. It is also seen in exophthalmic goitre. Tremor without known cause is sometimes hereditary. 4. Sudden *twitchings,* or a contraction of one or more muscles, may be due to direct motor irritation, or be of reflex origin in disease of the spinal cord. 5. *Fibrillary* contractions are due to spasm of separate fasciculi of the muscles. Such contraction is seen is spinal progressive muscular atrophy. 6. *Choreiform movements.* The movement may be slight and local, or general. It may be confined to the face or to an extremity. The movements are usually interrupted by pauses of irregular length. They occur in chorea and after hemiplegias. 7. *Athetosis.* (See page 131.) Slow involuntary movements, chiefly of the arm and hand, occur. They are of common occurrence in the cerebral paralysis of children.

8. *Coördinated spasms* are forced complicated movements, as spasms of jumping, laughing, running, moving in a circle, or turning about the axis of the body. They may be associated with spasm of the respiratory, pharyngeal, and laryngeal muscles. They occur in hysteria, certain forms of epilepsy, and disease of the cerebellum. 9. *Tonic spasms.* The muscles are in a constant state of rigidity, as the muscles of mastication in trismus. Muscles of the back and neck, when in tonic spasm, cause opisthotonos. 10. *Catalepsy.* The muscles remain in any position given to them on passive movement. They are deprived of the will. Catalepsy occurs in hysteria; rarely in meningitis.

Convulsions are divided into epileptiform or cerebral convulsions, in which consciousness is lost; hysterical convulsions, in which consciousness is disturbed, and spinal convulsions, in which consciousness is normal and reflex actions are exaggerated.

Ataxia. In ataxia, or incoördination, there is want of simultaneous action of muscles which are required to conduct complicated movements. Either there is (1) undue spasm, or (2) paralysis or paresis of one or more of the muscles involved in the complicated movement, or (3) the innervation of the muscles is abnormal, so that irregular contraction takes place in the production of the movement. The completion of a complicated act, as walking, is known as coördination. When the muscles do not act simultaneously incoördination is produced. Ataxia may involve all the muscles of the body or one of the extremities, so that we may have an ataxia of the arm, etc. Ataxia occurs in disease of the cerebellum and the spinal cord, as in locomotor ataxia (see page 60).

The Reflexes.

The reflexes are of two kinds, cutaneous reflexes and tendon reflexes.

Cutaneous Reflexes. When the sensory cutaneous nerves are irritated muscular contractions are excited in the vicinity. They are known as cutaneous reflexes. They are excited by pricking or pinching or by tickling the skin. The reflexes of the upper extremities are not marked. In the lower extremities they are more pronounced. They may be excited by tickling the soles of the feet, by pricking them with a pin, or by the application of ice to the skin.

In nervous diseases there is often *delay* in the reflexes, that is, no response to the irritation occurs until ten or fifteen seconds elapse. The reflex contractions are usually confined to the irritated limb. The irritability may be so great, however, as to cause a response from both legs or even the whole body, as in tetanus or strychnia poisoning. The following are special forms of cutaneous reflex: The abdominal reflex; the cremaster reflex (the scrotum is drawn up when the skin of the inside of the thigh is irritated); the gluteal reflex; the mammillary reflex, etc.

Even within the bounds of health variations in the reflexes occur in different individuals. If possible it is important to compare the reflexes on symmetrical portions of the body.

Absence of cutaneous reflex is seen in disease of the peripheral nerves and of the spinal cord, because the conduction of the reflex is inter-

rupted in its course. They are also absent when the reflex centres lose their irritability. Increase of the cutaneous reflexes occurs in strychnia poisoning, in cutaneous hyperæsthesia, and in general neuroses, because of increased irritability of the parts. In disease of the brain and spinal cord which causes degeneration of reflex centers or the inhibitory processes, they are abolished.

Tendon Reflexes. Muscular contractions occur from irritation of the tendons, the periosteum, or the fasciæ. The nerves of the tendon are irritated and excite reflex contraction.

The patellar reflex. This is detected when the patient crosses the leg loosely over the opposite knee, or when the limb is held up and hangs over the arm in a relaxed state. The tendon of the quadriceps muscle is struck by the finger or pleximeter. All muscular tension of the leg must be avoided. The reflex may be exaggerated by simultaneous muscular effort on the part of the patient, as contraction of a hand when the blow is given.

Ankle clonus. When the tendo Achillis is made tense by a short, vigorous, dorsal extension of the foot the reflex is exaggerated, plantar flexion of the foot taking place. If persistent dorsal extension of the foot be applied the foot is put into a vigorous tremor. Other reflexes are obtained in the lower extremities. They are elicited by a blow on the periosteum or fasciæ, etc.

The tendon reflex is absent in poliomyelitis, locomotor ataxia, and peripheral neuritis. It is increased in cerebral and spinal paralysis.

Vasomotor, Trophic, and Secretory Disturbances. 1. Vasomotor paralysis. This is indicated by abnormal redness of the skin, with increase in the temperature and a sensation of heat. They occur in functional neuroses, as hysteria and neurasthænia, and follow injuries of the sympathetic nerve. (See Hyperæmia, p. 172.)

2. Vasomotor spasm. There is pallor and coolness of the skin, because of spasm of the small vessels. There is formication and stiffness. It is most common in the hands. It may give rise to trophic disturbances, as in symmetrical gangrene, scleroderma, and similar diseases.

The following trophic disturbances described elsewhere are symptoms of functional or organic disease: 1. Angio-neurotic œdema. 2. Herpes zoster. 3. Urticaria. 4. Atrophy of muscles and nerves. 5. Atrophy of the skin (see Glossy Fingers). 6. Acute bedsores. 7. Myxœdema. 8. Trophic changes in the skin, nails, and hair. 9. Acromegalia. 10. Trophic swellings of the joints.

Electrical Diagnosis.

For purposes of diagnosis we use two forms of current—the faradic and the galvanic. Practically we study the reaction of the muscle to stimulation through its nerve, for we cannot, except in experimental work upon animals, in which nerve and muscle can be isolated, limit the action of the current to a muscle without influencing the nerve fibres in it. In testing a nerve it will be found that the result varies according to the current used. Thus, if the two poles of a faradic battery be applied, say, to the ulnar nerve, there results a muscular contraction, the

presence and force of which depends upon the strength of the current and not upon which pole is directly over the sensitive point. With the galvanic current the matter is more complicated. If one large electrode is placed at some indifferent point, say on the sternum or between the scapulæ, and the other smaller electrode over the nerve, and the current passes, it will be found that so long as the current is not interrupted no contraction of the muscle will occur. If, however, the moderately strong current be interrupted there will appear at each opening and closing of it a contraction. But the presence of a contraction depends upon which pole is over the nerve and whether the current is opened or closed. That is, the strength of current needed to produce a contraction varies according to whether the positive or negative pole is over the nerve and whether the current is opened or closed; in other words, if we begin with a very weak current there is no contraction under any circumstances, and in slowly increasing it we find that the first contraction occurs when the negative pole is over the nerve and the current closed. At the moment of opening the current, and when it passes without interruption, there is no contraction. If the current is increased still more, the contraction at closing—the negative pole still being over the nerve—becomes stronger, and as the current is increased contraction will occur when the positive pole is over the nerve. In this case contraction usually appears first at the opening and later at the closing. If the current be still further increased we obtain a contraction at the opening, the negative pole being over the nerve. In order to obtain this reaction the current may need to be so strong as to be painful. This, then, is the order in which contractions occur in the healthy nerve and muscle. We can make it more easily understood by the following formula. Let A. represent the positive pole or anode, C. the negative pole or cathode, O. the opening, and C. the closing. Thus:

Negative closing = C. C.
Positive opening = A. O.
Positive closing = A. C.
Negative opening = C. O.

Any deviation from this formula denotes disease. For instance, if C. O. contraction occurs with the same strength of current as C. C. contraction it would be conclusive evidence of some pathological change in the nerve or trophic centres. In certain diseases we find distinct and definite changes in their reactions. Let us take, for instance, the peroneal nerve in a case of acute anterior poliomyelitis. For the first few days after the onset of the disease there will be a diminishing response of the nerve to both faradic and galvanic currents. If the current be applied directly over the muscle it will be found that the response to faradism rapidly decreases and finally may be entirely lost, while the response to galvanism is not nearly so much diminished, and may, toward the end of the second week, actually increase. Not only do we have an increase but the polar reaction is changed, so that A. C. contraction may equal C. C. contraction, and after a while C. O. contraction may equal A. O. contraction, or C. O. contraction may appear with a less current than A. O. contraction. The character of the contractions varies also. In the healthy muscle they are quick, shock-like; in the diseased,

sluggish and worm-like. These alterations constitute the reaction of degeneration (De. R.). Finally, the muscle may cease to respond to the galvanic current no matter how strong it may be.

The reaction of degeneration often requires much skill to determine, and as it is probably never present in a muscle which contracts normally to the faradic current the failure of response to this current is the best test for the practitioner as to the condition of the muscle and nerve. The presence of reaction of degeneration means that the lesion is either in the nerve trunk (neuritis), the anterior motor cells of the cord (poliomyelitis), or the nuclear origin of a nerve. It is never caused by a cerebral lesion. In purely muscular diseases, as, for example, pseudomuscular hypertrophy, there may be diminution or absence of electrical response but never reaction of degeneration.

Cerebral and Spinal Localization.

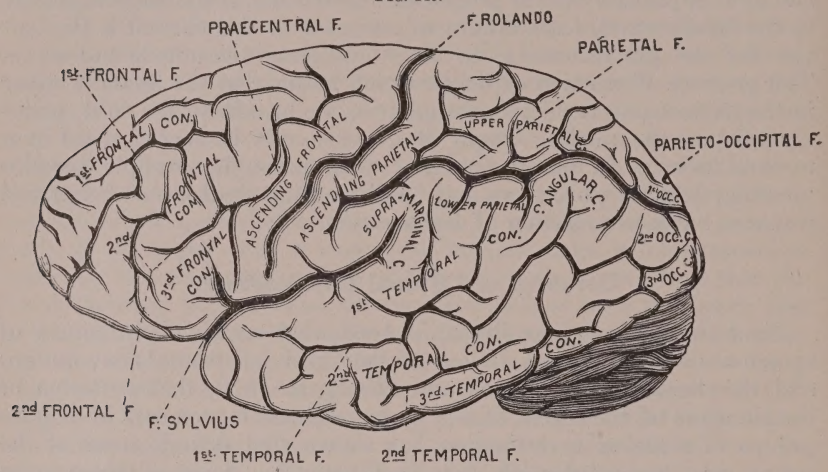
Since the discovery by Broca, in 1861, that certain disturbances of speech are associated with lesions of the third left frontal convolution, and the discovery by Fritsch and Hitzig, in 1870, that irritation of certain areas of the cortex of the brain produces movements in definite groups of muscles, investigation has shown that definite areas of the cortex are concerned with definite functions. Some of these areas (centres) are now well known and their localization determined, and it is the purpose of the present chapter to study the symptoms found in diseases of them. The position of a lesion, then, is determined by the symptoms; but all symptoms are not of equal localizing value, some indeed being valueless. All symptoms are due either to destruction or irritation of nerve tissue, and both occur in every lesion. The former, called "direct," are permanent unless some other part assumes the function of the part destroyed. The latter, called "indirect," are transitory unless the lesion be a slowly increasing one, in which case, as for example a tumor, they recur but do not persist. "Indirect" symptoms are produced by changes in circulation and compression around the focus of disease. We must wait for them to pass away before attempting to localize the lesion. Again symptoms are "focal" or "diffuse." The former are due to interference with the function of some definite part of the brain, while the latter may be caused by disease in any position. The commonest "diffuse" symptoms are headache, vomiting, loss of consciousness, and optic neuritis. The value of "focal" symptoms in localization depends upon whether they occur only when the lesion is in one definite area or in one of several. If the onset is acute it is necessary to know that all the symptoms appeared at the same time, as otherwise they must have been caused by different lesions. In a chronic but progressive disease there is, of course, gradual increase of the symptoms.

Cerebral Localization.

Cerebral Cortex. The *motor area* includes the ascending frontal, the parietal, and the posterior portion of the frontal convolutions and the paracentral lobule. The upper third contains the centre for the leg of

the opposite side, the middle third that for the arm, and the lower third that for the head and neck. The centre for the motor mechanism of speech is in the third left frontal convolution.

FIG. 149.



Convolutions of the left hemisphere. (L. C. GRAY.)

Destructive lesions in this area cause paralysis of one limb (monoplegia), or of a group of muscles. In order that all the centres should be affected and palsy of the opposite half of the body (hemiplegia) ensue, the lesion would have to be so great as, in an acute disease, to cause

FIG. 150.

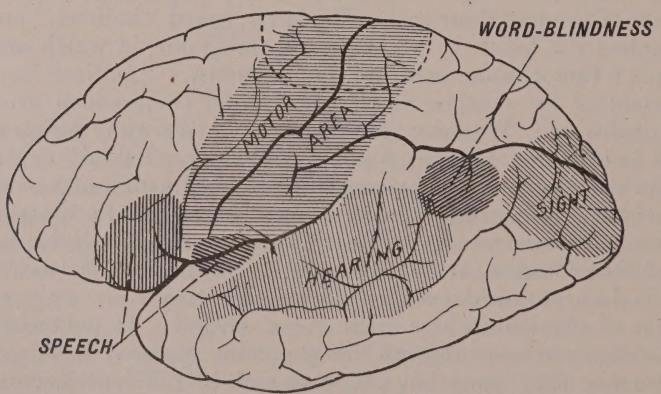


Diagram showing localization of centres in the cortex. (L. C. GRAY.)

immediate death. If, however, the lesion is not confined to the gray substance but penetrates the white matter, fibres from healthy portions of the cortex may be interrupted and a more extensive palsy result than is found in a purely cortical lesion. Further, a minute lesion of the white matter may, as shown below, produce a monoplegia, and there-

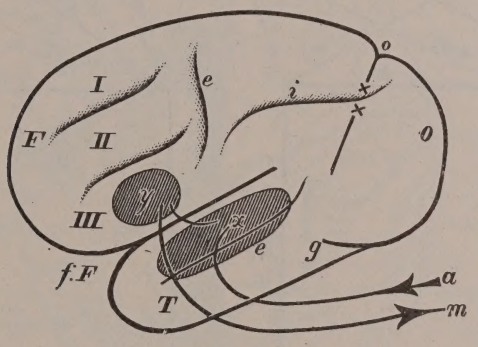
fore, palsy of one extremity does not prove absolutely the presence of cortical disease.

Irritative lesions cause convulsion in the muscles controlled by the affected part. The convulsions, however, are often not limited to the muscles in which they arise, but extend throughout one side or over the whole body. Again, while several centres may be diseased, the convulsion may always start in one limb or group of muscles. In general, we may say that convulsion is of less localizing value than paralysis, because in the latter case the seat of disease must be in the centre itself, or in the fibres from it, while in the former it need only be near the centre. General convulsions are of course of no localizing value.

Pre-frontal Lobe. Lesions of the frontallo be anterior to the motor area produce either no symptoms at all or purely mental ones, and hence it was formerly held that this area was the seat of the mind. It is now largely held, however, that mind is an attribute of the entire cerebral cortex, and it is certainly true that lesion in any part may, if extensive enough, produce mental symptoms.

Cortical Centres of Speech (*Aphasia*). The speech centres are situated in the third left frontal and first temporal convolutions in right-handed

FIG. 151.



Wernicke's schema for the cortical mechanism of speech.

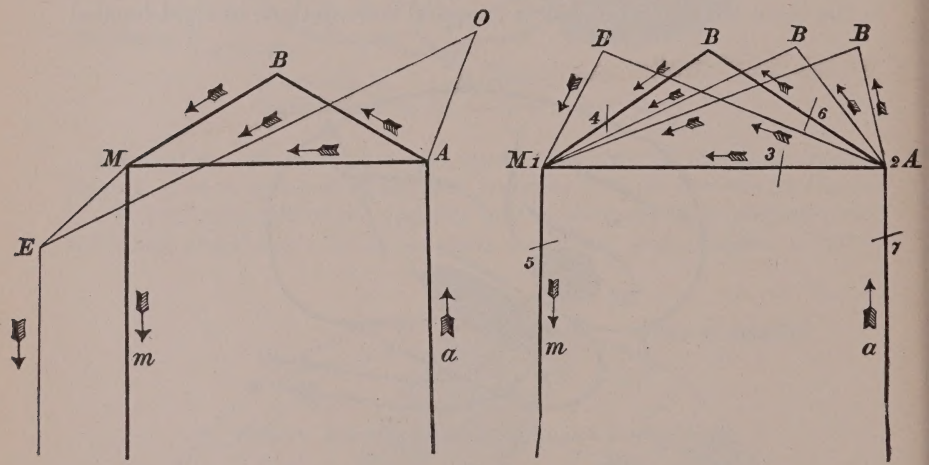
people, while curiously enough in left-handed people the centres are usually if not always upon the right side. Lesion of them or of the association path between them situated in the insula(?) results in different forms of affection of speech called collectively aphasia. It must be remembered that aphasia is not due to a paralysis of the muscles of articulation and phonation, but to a mental inability to select the proper word or to determine the necessary movements for its pronunciation.

In the accompanying diagram from Wernicke the motor (*y*) and sensory (*x*) speech centres are represented. If the lesion is at *y*, *motor aphasia* results. There is no palsy of the muscles used in speech, and the patient hears perfectly and knows what he wishes to say, that is, he has perfect recollection of words, yet he cannot speak at all or can only say a few words or syllables. There is often inability to write (agraphia) without paralysis of the hand or mind-blindness, and sometimes inability to read (alexia).

If the lesion is at x, which is the termination of the centripetal path of the auditory nerve, "sensory aphasia," "word-deafness," results. The power of hearing sound is preserved, but the ability to interpret the meaning of heard words is lost. If the lesion is absolute the patient is unable to repeat heard words. He may have as large a vocabulary as ever, but he makes mistakes both in the words used and in their form. The errors are especially marked in the voluntary revival of words, while automatic speech, as in singing or swearing, may be normal. Nouns are more apt to be lost than verbs, adjectives, and prepositions. Circumlocution is often used—for example, the patient may say "that with which one cuts," meaning "knife."

If the lesion is between y and x in the insula(?), "amnesic" or "conduction aphasia," results. In this case there is no loss in the motor speech processes nor word-deafness, but there is difficulty in recalling words, and they are used improperly. If both x and y be involved there is "total aphasia." The patient loses both power and understanding of speech.

FIG. 52.



Schema illustrating the seven different forms of aphasia. *a A*, centripetal path for auditory impressions; *A*, centre for auditory images; *M*, centre for motor images; *Mm*, centrifugal motor path; *B*, the place where concepts are formed; *O*, the centre for visual images; *E*, the centre from which the organs of writing are innervated. (LICHTHEIM.)

If the lesion is in the supra-marginal and angular convolutions, "alexia," "word-blindness," results. The patient cannot recall the appearances of words and does not recognize print or writing. He may be able to pronounce letters and can often write correctly, but cannot read understandingly what he has written. Word-blindness is a part of the larger symptom "apraxia," mind-blindness, in which the patient, while seeing objects, fails to recognize their nature and characteristics by vision.

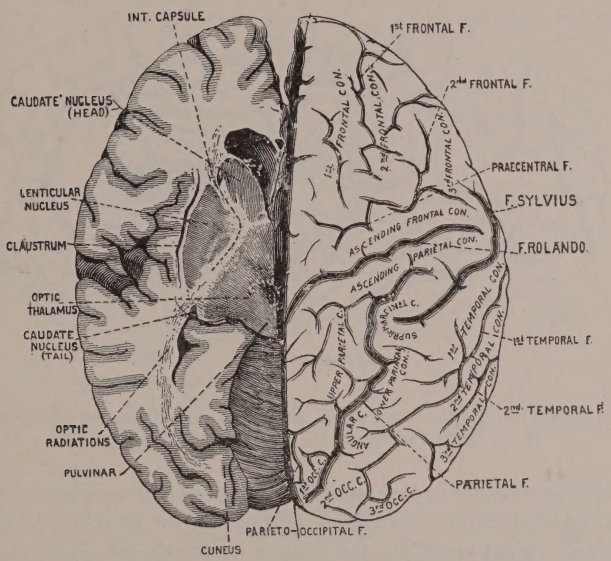
A study of the above diagram from Lichtheim will probably make the whole subject clearer.

In apraxia the concept centre (B) is affected; in motor aphasia the

lesion is at *m*; in sensory aphasia the lesion is at *A*; in alexia the lesion is at *O*; in conduction aphasia the lesion is somewhere in the path connecting *A M* and *O M*. In every case of suspected aphasia the following tests should be made: 1. Ability to recognize the nature and uses of objects. 2. Ability to recall the names of things seen, smelled, tasted, touched, or heard. 3. Ability to understand spoken words. 4. Ability to understand printed or written words. 5. Ability to understand musical tunes. 6. Power of voluntary speech. 7. Ability to read aloud and understand what he reads. 8. Ability to write and understand what he has written. 9. Ability to copy writing or print. 10. Ability to write at dictation. 11. Ability to repeat words heard.

It must be remembered that by the bedside the problem is much more complex than appears here. The cases are often not clearly separated, but various types run into each other, and the severity of the symptoms varies greatly.

FIG. 153.



Convolutions of the vertex, on the right; on the left, the basal ganglia, internal capsule, centrum ovale, and the cuneus. (L. C. GRAY.)

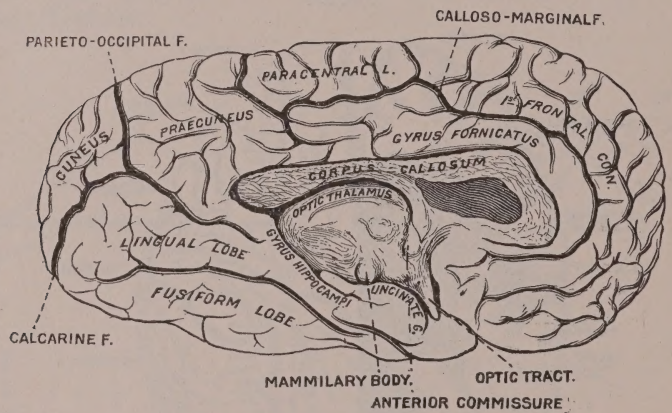
Parietal Lobe. Extensive disease probably interferes with sensation on the opposite side of the body. The functions of the ascending parietal and of the paracentral lobules have already been described.

Occipital Lobe. The cortical centre of vision is in the cuneus and the adjacent convolutions. Disease in it produces hemianopsia which is described under a special heading.

Corpus Callosum. No localizing symptoms occur in disease in this region. Mental dulness and bilateral weakness result sometimes from tumor. The *centrum ovale* contains fibres from the cortex which come closer together and occupy a smaller and smaller space until the internal

capsule is reached. It follows, therefore, that the nature of the symptoms will vary with the distance of the lesion from the cortex. If near the cortex the symptoms must resemble those found in corresponding cortical disease, while if near the internal capsule they will in turn resemble those found in disease there. Thus a lesion under one of the motor centres will produce a monoplegia, while if deeper a hemiplegia will result. Local convulsions occur only when there is an irritative lesion immediately below the cortex, and general convulsions only in disease causing increase of cerebral pressure, as, for example, tumor. If the lesion is extensive there may be hemianæsthesia on the opposite side. Disease of the white matter of the occipital lobe may cause hemianopsia; of the temporal lobe, auditory disturbance. The differential diagnosis between a cortical and subcortical lesion is often difficult and sometimes impossible.

FIG. 154.



Vertical section through the centre of the corpus callosum, showing the convolutions of the median surface of the hemisphere. (L. C. GRAY.)

The *internal capsule* is the most frequent seat of cerebral disease, the lesion being most often vascular—embolism, or rupture of an artery. If the lesion is situated in the anterior third between the caudate nucleus and the lenticular nucleus, so far as known no definite symptoms result, but if it is in the middle third we have hemiplegia of the common type. The lower face, the tongue, the arm, and the leg on the opposite side are all affected, and if the palsy be right-sided there is at the beginning defect of speech. There may also be at the first deviation of the head and eyes, but never permanent palsy of any cranial nerve. Later on rigidity develops in the muscles, the knee-jerk is increased and ankle-clonus appears. Sometimes the hemiplegia is not complete, for if the lesion is small many fibres may escape, but there is practically never a true monoplegia. The sensory fibres from the cortex pass through the posterior third, and consequently if it be involved hemianæsthesia results, and there may be hemianopsia and loss of smell on the anæsthetic side.

The Corpus Striatum and Optic Thalamus. Lesions of the basal ganglia give no diagnostic symptoms unless the internal capsule is involved.

The Corpora Quadrigemina are closely connected with the optic-nerve fibres, the tegmentum, the superior and middle cerebellar peduncles, the pineal gland, the pulvinar, and the nuclei and fibres of the ocular nerves. In consequence, disease in this region is accompanied by numerous and varying symptoms. Ataxic gait, similar to that present in cerebellar disease, ophthalmoplegia, and nystagmus are somewhat characteristic symptoms.

FIG. 155.

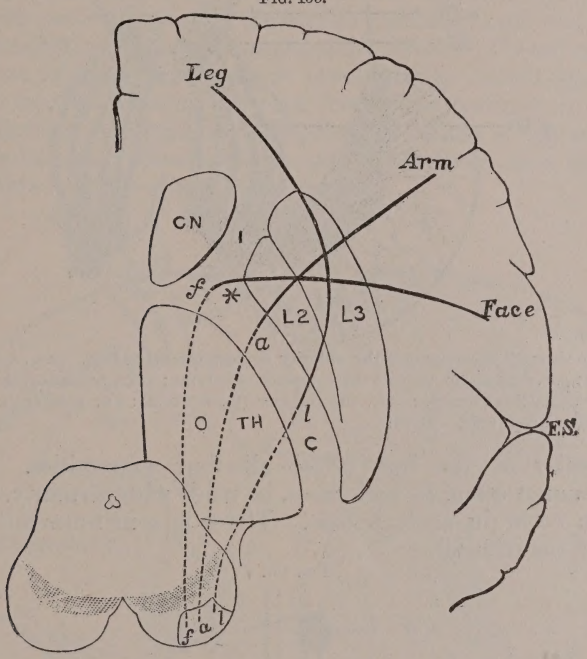


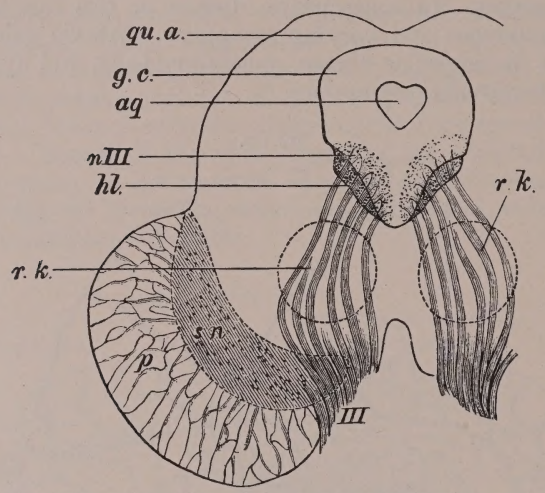
Diagram to show the relative position of the several motor tracts in their course from the cortex to the crus. The section through the convolutions is vertical; that through the internal capsule, I C, horizontal; that through the crus is again vertical. C N, caudate nucleus; O TH, optic thalamus; L 2 and L 3, the middle and outer parts of the lenticular nucleus, *f a l*, face, arm, and leg fibres. The words in italics indicate the corresponding cortical centres. (GOWERS.)

The Crus Cerebri is in close anatomical relation with the oculo-motor nerve, as is shown in the diagram. We find, therefore, characteristic symptoms in disease. There is always oculo-motor palsy on the same side as the lesion and hemiplegia on the opposite side, both coming on at once. If there is anæsthesia on the palsied side the tegmentum is also involved.

Pons. The symptoms depend upon the level at which the lesion is situated. The fibres of the facial nerve decussate higher up than those of the pyramidal tract, and consequently a lesion in the lower part will cause facial palsy on the same side and palsy of the leg and arm on the opposite side (alternating paralysis). If the lesion is above the facial nerve decussation there will result hemiplegia of the opposite side,

including the face—distinguishable, however, from the typical hemiplegia from disease of the internal capsule by the fact that all the branches of the facial are affected, and that there may be, though rarely, reaction of degeneration. Bilateral lesions may cause bilateral facial palsy, or

FIG. 156.



Cross section through the region of the anterior corpora quadrigemina. *qu.a.*, anterior corpora quadrigemina; *g.c.*, gray matter around the aqueduct of Sylvius; *nIII*, nucleus of the third nerve; *hl*, posterior longitudinal bundle; *r.k.*, red nucleus (tegmentum): *s n*, substantia nigra (locus niger); *p*, cerebral peduncle. (HIRT.)

bilateral palsy of the legs or of all four extremities. The local diagnosis cannot often, in such cases, be made with certainty. Convulsions often occur in acute lesions. There is sometimes anæsthesia in the area of the trifacial.

FIG. 157.

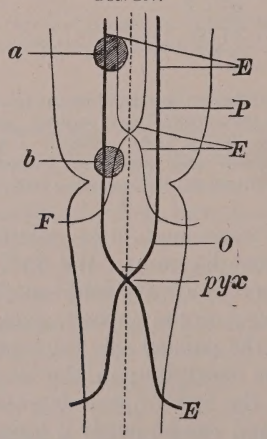


Diagram showing the decussation of the fibres going to the extremities, and of those going to the face in the pons and medulla oblongata. *F*, facial fibres; *E*, fibres going to the extremities; *P*, pons; *O*, medulla oblongata; *pyx*, decussation of the pyramidal tracts; *a*, a focus in the upper; *b*, a focus in the lower part of the pons (the latter is situated below the decussation of the facial fibres). (HIRT.)

Cerebellum. The hemispheres may be extensively diseased without giving rise to any symptoms. The characteristic symptom of disease of the middle lobe is disturbance of equilibrium and incoördination. The gait resembles that of a drunken man. Giddiness and vomiting sometimes occur, but are of no localizing value. Nystagmus is frequent in cases of tumor. The knee-jerk is often absent, or it may be sometimes absent and sometimes present. If the pyramidal tracts are pressed upon, the reflexes are increased and there is weakness in the corresponding extremities. There may be palsy of the cranial nerves, difficulty in articulation due to pressure on the medulla, and occasionally epileptiform convulsions. If the middle peduncle is affected by an irritative lesion, quite characteristic symptoms result. "Forced movements" occur—that is to say, the body is involuntarily rotated upon its long axis, and the patient may have an irresistible tendency to lie on one side. There are no diagnostic symptoms of disease of the superior and inferior peduncles. Disease of one side of the pons may cause symptoms similar to those of cerebellar trouble.

FIG. 158.

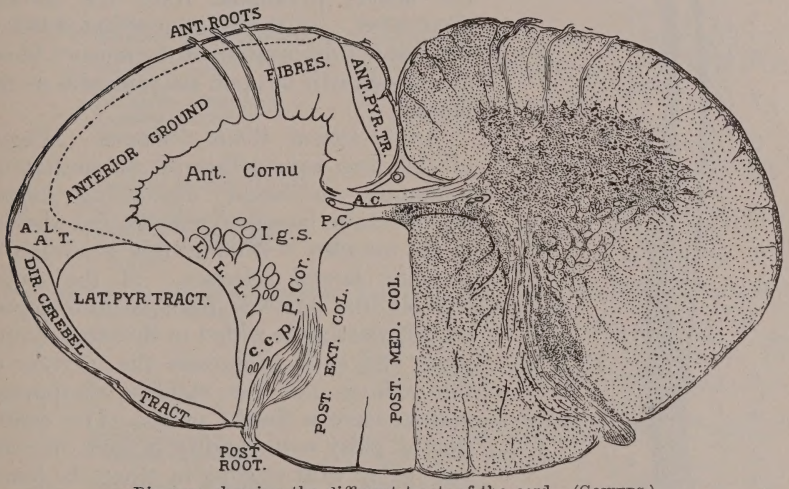


Diagram showing the different tracts of the cord. (GOWERS.)

Medulla Oblongata. If the nuclei in the floor of the fourth ventricle are diseased, bulbar palsy, which is described on page 844, results. It must be remembered that bilateral lesions in the lowest part of each ascending frontal convolution may cause symptoms indistinguishable from those of true bulbar palsy.

Spinal Localization.

The localizing symptoms in disease or injury of the cord vary with the level at which the lesion is situated and with the part of the transverse area involved.

A total transverse lesion causes, of course, total paralysis of all parts below, including the bladder and rectum, with anæsthesia. If

situated above the lumbar enlargement, the knee-jerk is increased, the legs become spastic, and ankle clonus appears on account of secondary degeneration of the lateral tracts. If the lumbar enlargement is involved the reflexes are abolished and the palsy is flaccid. Much finer local diagnosis can be made by a study of Fig. 159, and of the table of the functions of the different segments of the cord which I quote from M. Allen Starr. It is important to remember that the segments of the cord do not correspond to the vertebræ after which they are named.

FIG. 159.

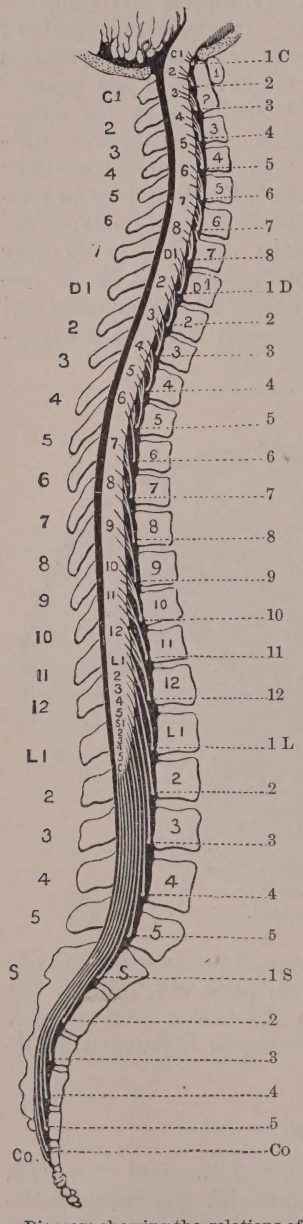


Diagram showing the relations of the vertebral bodies and spines to the segments of the cord and to the exit of the nerves. (GOWERS.)

Unilateral lesions produce palsy on the same side, with increased reflexes and rigidity, and on the opposite side anæsthesia, reaching not quite up to the seat of lesion. There may be some palsy on the side opposite the lesion on account of some fibres of the lateral pyramidal tract not having decussated. If the lesion is situated below the point of decussation of the sensory fibres anæsthesia will be upon the same side as the palsy.

Antero-lateral White Columns. Disease of this area causes loss or diminution of voluntary movement, descending lateral degeneration, increased reflexes, and rigidity of the muscles. We find this condition in primary lateral sclerosis. If the motor cells in the anterior horn be also degenerated, wasting is added to the other symptoms, and as it progresses the increase of the reflexes and the rigidity disappears, and we have a flaccid palsy. This condition of palsy with rigidity in some muscles and palsy with wasting in others is found in amyotrophic lateral sclerosis.

Posterior White Columns. If the postero-external columns be affected there results muscular incoördination, with no loss of power, lancinating pains, abolished knee-jerk, and impaired sensation. Locomotor ataxia is the type of disease in this region. The symptoms of disease of the postero-median columns are unknown.

Anterior Horns. The large cells in the anterior horns are the trophic cells of the nerves proceeding from them. Disease of them, therefore, is followed by muscular wasting. There is also, of course, palsy,

Segment.	Muscles.	Reflex.	Sensation.
II. and III. C.	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	Hypochondrium(?). Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.
IV. C.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra-spinatus.	Pupil. 4th to 7th cervical. Dilatation of the pupil produced by irritation of neck.	Neck. Upper shoulder. Outer arm.
V. C.	Deltoid. Biceps. Coraco-brachialis. Supinator longus. Supinator brevis. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular. 5th cervical to 1st dorsal. Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator longus. Tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm, front and back.
VI. C.	Biceps. Brachialis anticus. Pectoralis (clavicular part), Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps. 6th to 7th cervical. Tapping elbow tendon produces extension of forearm. Posterior wrist. 6th to 8th cervical. Tapping tendons causes extension of hand.	Outer side of forearm, front and back. Outer half of hand.
VII. C.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Anterior wrist. Tapping anterior tendons causes flexion of wrist. Palmar. 7th cervical to 1st dorsal. Stroking palm causes closure of fingers.	Inner side and back of arm and forearm. Radial half of the hand.
VIII. C.	Flexors of wrist and fingers. Intrinsic muscles of hand.		Forearm and hand, inner half.
I. D.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.		Forearm, inner half. Ulnar distribution to hand.
II. to XII. D.	Muscles of back and abdomen. Erectores spinæ.	Epigastric. 4th to 7th dorsal. Tickling mammary region causes retraction of the epigastrium. Abdominal. 7th to 11th dorsal. Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen, in bands running around and downward corresponding to spinal nerve. Upper gluteal region.
I. L.	Ilio-psoas. Sartorius. Muscles of abdomen.	Cremasteric. 1st to 3d lumbar. Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum.
II. L.	Ilio-psoas. Sartorius. Flexors of knee (Remak). Quadriceps femoris.	Patellar tendon. Striking tendon causes extension of leg.	Outer side of thigh.
III. L.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.		Front and inner side of thigh.
IV. L.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier). Tibialis anticus.	Gluteal. 4th to 5th lumbar. Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
V. L.	Outward rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle. Extensors of toes.		Back of thigh, back of leg, and outer part of foot.
I. to II. S.	Flexors of ankle. Long flexors of toes. Peronei. Intrinsic muscles of foot. Perineal muscles.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg. Foot reflex. Achilles' tendon. Over-extension of foot causes rapid flexion; ankle clonus. Bladder and rectal centres.	Back of thigh. Leg and foot, outer side. Skin over sacrum. Anus. Perineum. Genitals.

because these cells are, if one may say so, a way-station between the periphery and the cortex. And as they also are a link in the reflex arc, the muscle reflexes are abolished. Wasting is the most important symptom, for from it we can say positively that the anterior horns are affected. A short time after the onset, too, reaction of degeneration appears in the muscles. There may be slight wasting in cerebral palsy, but it is simply from disuse, and reaction of degeneration is never present. The extent of the palsy and wasting depends upon the extent of cord involved. It may be monoplegic, or several or all extremities may be affected.

Diseases of the Cranial Nerves. Examination of the Functions of the Cranial Nerves.

Olfactory Nerve. A rhinoscopic examination should always be made to discover whether local disease exists, since this may destroy all sense of smell. Irritant substances must not be used in examining, since they stimulate the trifacial nerve. Oil of cloves and peppermint or any of the essential oils are the best. Each nostril should be examined separately. Disturbance of function may arise from a lesion anywhere between the periphery and the cortical origin.

Anosmia, loss of the sense of smell, may be caused by acute or chronic nasal catarrh ; abnormal dryness of the mucous membrane from disease of the trifacial ; traumatism of the bulbs or nerves ; meningitis or tumor causing pressure upon or inflammation of the bulbs or nerve trunks ; and finally, lesions of the olfactory centre, placed by Ferrier in the uncinate gyrus. It is frequently met with in hysteria, and is sometimes seen in workers in strong-smelling substances.

Parosmia, subjective sensation of smell, is found among the insane and in cases of migraine, tic douloureux, epilepsy, hysteria, and tabes dorsalis. Usually the odors are unpleasant.

Hyperosmia, increased acuteness of smell, occurs in hysteria. It may be so marked as to make it possible for the patient to recognize persons by smell alone.

Optic Nerve and Tract. This is the most important of the cranial nerves in relation to general diseases, especially those of the nervous system.

1. *Retinal Lesions.* (*a*) Retinitis sometimes occurs as an idiopathic affection, but more frequently it is found in association with Bright's disease, syphilis, leukæmia, and severe anæmia. It occurs occasionally in diabetes, purpura, and chronic lead-poisoning. Whatever the cause, there are seen on ophthalmoscopic examination of the retina white spots and patches of various sizes and distribution, due for the most part to degenerative processes and hemorrhages. The latter are in the nerve-fibre layer and often follow the course of bloodvessels. When recent they are bright red in color, when old, black.

Albuminuric retinitis occurs only in chronic renal disease. It is most frequent in granular kidney, least so in lardaceous disease. It may be present when there is little or no albumin in the urine, and practically

is never coexistent with functional albuminuria. Its presence proves organic renal disease. Gowers distinguishes four types—the degenerative, the hemorrhagic, the inflammatory, and the neuritic, according as white spots of degeneration, extravasations of blood, parenchymatous retinal inflammation, or inflammation limited to the optic nerve predominates.

b. Functional blindness, toxic amaurosis, occurs quite often in uræmia, sometimes in acute or chronic lead-poisoning, and occasionally from quinine. In hysteria there may be blindness in one or both eyes, but more often there is only a marked decrease in visual acuity. In this condition ophthalmoscopic examination reveals nothing.

Tobacco amblyopia is gradual in onset and equal in both eyes. It is characterized by defect in the centre of the field of vision, a central scotoma. The scotoma is relative, not absolute; vision is dimmed, not lost, and the failure is greater for red and green than for white. The eye-grounds may be normal, but if tobacco be persistently used, atrophy of the disk may result.

Nyctalopia, or night-blindness, is the condition in which objects are clearly seen in a bright light, but are invisible in the shade or in twilight. In *hemeralopia* the reverse condition exists.

Retinal hyperæsthesia occurs in hysteria and rarely in retinitis.

Optic Neuritis. (Choked disk, papillitis.) The disk is swollen and hyperæmic, its edges are blurred, and a striated or grayish haziness spreads over its face and out upon the retina. If the swelling be great there is left a complete atrophy upon its subsidence. In the early stages there is often no disturbance of vision, and even when the inflammation is quite severe sight may for a considerable time be quite good.

Rarely papillitis is idiopathic. It occurs sometimes in anæmia and lead-poisoning, not uncommonly in Bright's disease as a neuro-retinitis, and commonly in meningitis and tumor of the brain. Its frequent presence in the last disease renders it of great diagnostic value. It must be remembered, however, that the presence of papillitis gives no information as to the locality or pathological nature of a brain tumor.

Optic Atrophy may result from alcohol, lead-poisoning, diabetes, and the specific fevers. Many cases are associated with spinal cord diseases, particularly locomotor ataxia. Secondary atrophy is most commonly the result of papillitis, but it may result from cortical brain disease or pressure on the chiasma or nerves.

The disk is pale or bluish or grayish, and its outlines are distinctly marked off. Visual acuity is lessened, color perception is altered, and the field is contracted. There is no pain and seldom photophobia. Prognosis is usually bad.

Diseases of the Optic Chiasm and Tract. There is a semi-decussation of the optic nerves at the chiasm. The fibres from the outer half of each retina pass to the centre on the same side, while those on the inner half cross and pass to the centre of the opposite side. Remembering this, the symptoms resulting from lesion of the chiasm or tract are easily understood.

Unilateral Lesion of the Tract. If the lesion is situated at, say, B, Fig. 161, there will result loss of function of the temporal half of the right and nasal half of the left retina, so that the patient sees objects only on the right side. This condition is called lateral or homonymous hemianopia.

FIG. 160.

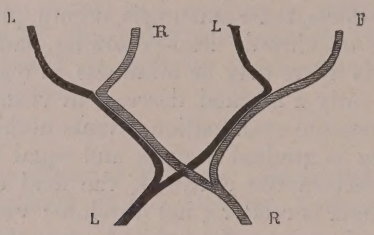


Diagram showing the course of the optic fibres in the chiasm. (HIRT.)

Lesion of the Chiasma. If the central portion of the chiasma, made up of decussating fibres from the nasal sides of the retinæ, be alone involved, there will result loss of vision in the outer half of each field—temporal hemianopia. If the lesion involves not only the central portion but also the direct fibres on one side, there results total blindness in one eye and temporal hemianopia in the other. Finally, if the entire chiasm is involved total blindness results. If the lesion affects the outer part of the chiasma, involving the fibres going to the temporal halves of the retinæ, blindness results in the nasal field—nasal hemianopia.

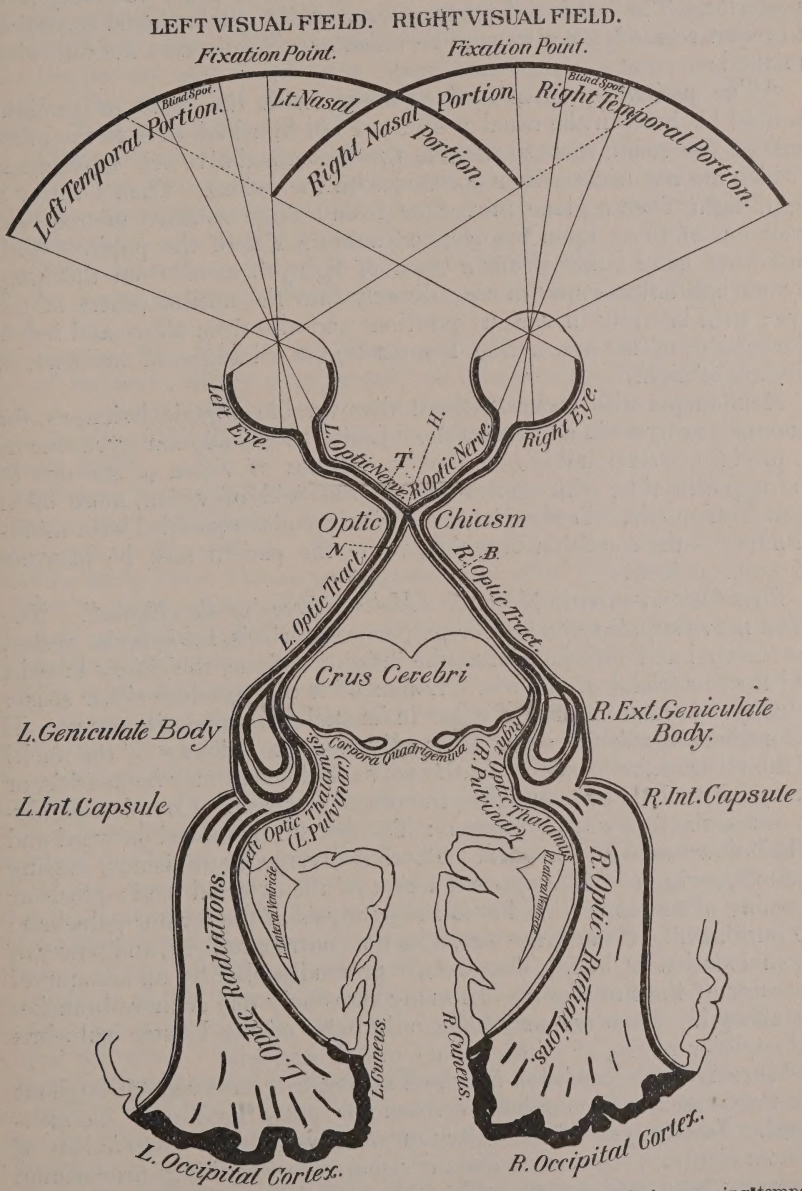
Lesion of the Tract and Centres. The optic tract, after crossing the crus to the hinder part of the optic thalamus, divides into two branches, one going to the thalamus and external geniculate bodies and to the anterior quadrigeminal bodies, from which fibres pass into the hinder part of the internal capsule, and, entering the occipital lobe, form the fibres of the optic radiation terminating in the cuneus, the perceptive visual centre, while the fibres of the other branch pass to the internal geniculate bodies and the posterior quadrigeminal bodies. It is held by some physiologists that the visual centre is not confined to the occipital lobe, but includes the occipito-angular region.

A lesion anywhere in the tract from the chiasma to the cortex will of necessity produce lateral hemianopia. To sum up, the lesion may be in the tract itself, in the region of the thalamus, in the corpora quadrigemina, in the fibres passing from the latter to the occipital lobe, either in the hinder part of the internal capsule, or in the white fibres of the optic radiation, or finally in the cuneus. Bilateral disease in any of these situations will of course produce blindness in both eyes.

Can we locate more closely the seat of the lesion? In some cases, yes. If it is in the posterior part of the internal capsule, hemianæsthesia of the opposite side, and if it extends far enough forward in the capsule, hemiplegia, will be associated with the hemianopia. Again, it has been found by Wernicke that if a beam of light is thrown laterally into a hemianopic eye, in a certain proportion of cases, on the blind side, the pupil will not contract. Now the light-reflex of the

pupil depends upon the integrity of the retina, of the fibres of the nerve and tract, and of the nerve centre in the geniculate bodies which receives the impression and transmits it to the third nerve, along which

FIG. 161.



The optic and visual tracts. *N*, lesion causing nasal hemianopia; *T*, lesion causing temporal hemianopia; *H*, lesion causing bilateral heteronymous hemianopia; *B*, lesion of tract causing homonymous hemianopia. (STARR.)

the motor impulse passes to the iris. If then we find, on examining the pupil by the method detailed below, that there is pupillary inaction on the side corresponding to the blind half of the retina, we are justified in saying that the lesion is very probably in the geniculate bodies or anterior to them, while, if both sides of the pupil respond, it is posterior. The test is a delicate one, not easily obtained, and according to recent research not always to be relied on. Seguin uses the following method:

"The patient being in a dark room, with the lamp or gaslight behind his head in the usual position, I bid him look over to the other side of the room, so as to exclude the accommodative iris movements (which are not necessarily associated with the reflex). Then I throw a faint light from a plane mirror or from a large concave mirror held well out of focus upon the eye, and note the size of the pupil. With my other hand I now throw a beam of light, focussed from the lamp by an ophthalmoscopic mirror, directly into the optical centre of the eye; then laterally in various positions and also from above and below the equator of the eye, noting the reaction at all angles of incidence of the ray of light."

Hemianopia also occurs without discoverable organic lesion, as, for example, in hysteria and migraine. Lesion of the angular gyrus seems to produce crossed amblyopia, that is, dimness of vision of the eye of the opposite side, with contraction of the field of vision, more often than hemianopia. Lesions in this region are also associated with mind-blindness—the condition in which, while the patient sees, he does not recognize objects.

The Oculo-motor Nerve. *Motor Nerves of the Eyeball.* The third nerve supplies the levator palpebræ superioris, the superior rectus, the internal and inferior rectus, the inferior oblique, the ciliary muscle, and the constrictor of the iris. Lesion of it may produce either spasm or palsy. It may be affected either in its nucleus or along its course. If the nucleus be affected, there is usually associated disease of the nuclei of the other ocular nerves. In the nerve itself there may be neuritis, or it may be involved by meningitis, tumors, or aneurism. Complete paralysis causes the following symptoms: The eye can be moved outward and a little downward and inward. There is divergent strabismus, causing diplopia, owing to the unopposed action of the external recti; ptosis or drooping of the upper lid, due to the paralysis of the levator palpebræ; the pupil, while of moderate size, does not contract to light, and power of accommodation is lost. The eyeball protrudes slightly on account of the palsy of the three recti. In many cases only one or more branches are affected. Thus we may have only palsy of the levator palpebræ and superior rectus, or of the ciliary muscle and iris.

The remarkable condition in which at irregular intervals throughout life there recurs a complete oculo-motor palsy need only be mentioned. In paralysis of the ciliary muscle (cycloplegia) there is loss of accommodation, so that while distant vision is perfect things near cannot be clearly seen without the aid of convex glasses. The condition is frequently met with in diphtheritic palsy and in tabes.

The iris has three actions (Gowers): 1. Reflex contraction of the

sphincter on exposure of the eye to light. 2. Reflex dilatation by the radiating fibres on stimulation of a cutaneous nerve. 3. Contraction on accommodation, usually, but not necessarily, associated with convergence. There are, therefore, three forms of paralysis of the iris (iridoplegia):

1. *Accommodation*, in which during accommodation the pupil does not diminish in size. To test this condition, it is simply necessary to make the patient look at a distant object and then at a near one, both being in the same line of vision so as to avoid any change in the amount of light entering the eye.

2. *Reflex*, in which the pupil does not contract when exposed to light. Each eye must be examined separately, keeping the other covered, since light entering one eye acts on both pupils. It is best tested by having the patient look at a distant object in a darkened room, and then bringing a light suddenly in front of the eye. If the light reflex be lost and the accommodation reflex is retained, we call it Argyll-Robertson pupil.

3. *Loss of Skin Reflex.* Pinching or pricking the skin, say of the back of the neck, will in most healthy persons produce dilatation of the pupil, the afferent impulse being sent along the cervical sympathetic.

Ordinarily in iridoplegia the pupils are small, but they may be of medium size.

Nystagmus is a spasmodic condition of the muscles of the eye, producing rapid oscillations of the ball, usually horizontal, sometimes rotary, and rarely vertical. It is of little value as a symptom. It is found in many brain lesions, in albinism, and often in miners.

Blepharospasm, spasm of the orbicularis palpebrarum, may cause only a twitching of the eyelids, or it may be so severe as to forcibly press the eyelids together, so that the patient cannot open them.

Ptosis is of sufficient importance to require a more detailed description. It may be congenital, in which case it is apt to be bilateral and partial, due to lesion of the third nerve or its nucleus, as mentioned above; associated with cerebral disease without the other branches of third nerve being affected; or hysterical. When the cervical sympathetic is paralyzed, the upper lid on the same side is a little lower than the other, due to the palsy of the fibres of Müller. The movements of the lid are, however, unimpaired, and other symptoms of sympathetic palsy, such as contraction of the pupil, dilatation of the vessels of the surface, and altered secretion of sweat, are always present. Very rarely irritation of the fifth nerve will cause transient ptosis. Occasionally, especially in sickly women, there is a condition called *morning ptosis*, in which for some minutes after waking it is impossible for the patient to open the eyes.

The Trochlear or Pathetic Nerve. *Fourth Nerve.* The superior oblique muscle is supplied by the fourth nerve. Palsy of it causes defect in downward and inward movement, causing diplopia on looking downward.

The Trigeminus. The *Fifth Nerve* is the great sensory nerve of the face. It supplies the entire side of the face, the conjunctiva, the mucous membrane of the lip, gums, tongue, hard and soft palate, and of the

nose. It supplies the anterior two-thirds of the tongue with the nerves of taste. Its motor division supplies the muscles of the lower jaw, temporal, masseter, pterygoid, the mylo-hyoid, and the posterior belly of the digastric.

Paralysis is caused by (1) hemorrhage or other lesion in the pons; (2) tumor at the base of the brain, meningitis, or caries; (3) the branches may be affected in their course—the first by tumor pressing on the cavernous sinus, the second and third by tumor invading the spheno-maxillary fossa; (4) primary neuritis, which is very rare. Secondary neuritis by extension is of course common.

Symptoms. Sensory. Anæsthesia is present over the entire distribution or in one branch, according as the entire trunk or only one branch is affected. There is also loss of taste in the anterior two-thirds of the tongue, and of smell in the corresponding nostril. The salivary, lacrymal, and buccal secretions may be lessened. If the Gasserian ganglion is affected the eye inflames, the cornea becomes cloudy and may ulcerate. Herpes sometimes develops. The anæsthesia is apt to be preceded by darting, burning pain.

Motor. There is inability to masticate on the affected side. If the pterygoid be affected the jaw when depressed deviates toward the palsied side.

Spasm of the muscles of mastication produces *trismus.* It may be clonic or tonic and may occur in general convulsions, or, but this is rare, as an isolated affection. In the tonic form the jaws cannot be opened. Clonic spasm is exemplified in chattering teeth.

Gustatory. While as stated above there is apt to be loss of taste in the anterior two-thirds of the tongue, this is not always so. These fibres may escape or the lesion be situated within the pons when these fibres are separated from those of sensation.

Neuralgia. (Trifacial neuralgia, tic douloureux, prosopalgia.) The trifacial is more frequently the seat of neuralgia than any other nerve. All the branches are rarely affected together. The ophthalmic is most often affected, producing the so-called brow ague, *supra-orbital neuralgia.* The pain radiates from the supra-orbital notch over the anterior half of the head and may extend to the side of the nose. The supra-orbital notch, or the nerve just above it, is painful on pressure. In *ocular neuralgia* there is pain confined to the ball, or both eyes may be involved. In *infra-orbital neuralgia* the second branch is affected and the pain extends from the orbit to the mouth and over the cheek. There are points painful on pressure over the infra-orbital foramen, over the malar bone, and above the gum of the upper jaw.

If the third division be affected there is pain in the parietal region, the temple, the lower jaw, and the tongue. There are tender points at the inferior dental foramen, at the posterior part of the temporal region, and over the parietal eminence.

The pain is always severe and its description varies with the powers of the patient, burning, tearing, boring, etc. When sudden and severe the reflex muscular spasm, the *tic convulsif*, occurs.

The Motor Oculi Externus or Abducens. *Sixth Nerve.* The external rectus muscle is supplied by it. Palsy of it produces defect of

outward movement of the ball and consequent convergent strabismus. Diplopia occurs on looking to the paralyzed side.

Acute Ophthalmoplegia. Acute Nuclear Palsy. Sudden paralysis of all the ocular muscles sometimes occurs from hemorrhage in the region of the nuclei. The apoplexy is usually fatal. It is unknown whether multiple neuritis ever affects the ocular nerves. If it does, the resulting palsy would simulate nuclear disease.

Chronic ophthalmoplegia may affect either the external or the internal eye muscles. In the first the levator muscles and the superior recti are first affected, later the others, so that finally the balls are immovable and the eyelids droop. The condition may persist for years. It is often associated with general paralysis and tabes dorsalis. In the second there is no pupillary reflex either to light or with accommodation. The two forms may occur together.

Spasm of the Ocular Muscles. The varieties of spasm are grouped by Gowers into five classes:

1. *Associated Spasm from Central Disease.* In a paralyzing lesion of one hemisphere the eyes deviate toward this side because of the unopposed influence of the opposite hemisphere. An irritative lesion of one hemisphere causes conjugate deviation due to spasm toward the opposite side. It occurs at the onset of unilateral convulsions.

2. *Irregular Spasm from Brain Disease.* In irritating disease of the base of the brain, especially in meningitis, there may be spasm of one or more ocular muscles.

3. *Chronic spasm in individual muscles* is very rare except in cases of secondary deviation.

4. *Hysterical Spasm.* In fits of hysteria the eyes are usually directed upward and to one side, often concealing the cornea entirely, or there may be marked convergence, but never divergence. Convergence may persist during the interval.

5. *Paroxysmal spasm* occurs in convulsive attacks. Cases are occasionally met with in which only one muscle is affected, there being at the same time momentary loss of consciousness.

The Facial Nerve. *The Seventh. Paralysis* may result from lesion of the cortex, of the nucleus, or of the trunk. The cerebral form, that due to disease above the nucleus, is easily distinguished from the peripheral, Bell's palsy, by the persistence of normal electrical reactions in muscles and nerves, and by the non-involvement of the upper branches, so that the orbicularis palpebrarum and frontalis are spared. Again, voluntary movements are more impaired than emotional ones. Ordinarily also supra-nuclear disease causes not palsy of the face alone but a hemiplegia.

Nuclear palsy rarely occurs alone, but is seen sometimes in tumors, chronic softening, and hemorrhage. In anterior poliomyelitis and diphtheria the nucleus may be affected. The condition cannot be diagnosticated from disease of the trunk.

In the peripheral form, Bell's palsy, all branches of the nerve are involved. The face on the affected side is motionless, the skin is smooth, the eye cannot be closed, and the lower lid droops. The angle of the mouth droops and the lips on the affected side cannot be closed.

On movement the face is strongly drawn to the sound side. The patient cannot whistle, and he may have difficulty in pronouncing the labials. Food collects between the teeth and the cheeks. The tongue appears to be protruded to one side on account of the facial deformity. In the great majority of cases the uvula does not deviate. All reflex movements are lost. There is no change in the electrical reactions in slight cases. In severe ones degeneration reaction is found. If the lesion is situated between the geniculate ganglion and the origin of the chorda tympani there is loss of the sense of taste in the anterior part of the tongue on the affected side. Hearing may be impaired usually on account of preceding ear disease. In old cases there occurs a secondary contraction of the muscles on the affected side, which draws the face to that side, increases wrinkling, and makes it appear while at rest that the diseased is the healthy side.

Double facial palsy is very rare, but may be caused by lesions at the base, in the pons, by disease in both ears, and possibly by disease of the nuclei or double cortical lesions.

Spasm may involve a few or all of the muscles and may be bilateral. If the muscles around the eye be affected it is called blepharospasm. More often there is twitching of all the muscles of the side of the face and partial closure of the eye.

The Auditory Nerve. *The Eighth. Hearing* is tested by the watch or a tuning-fork. Normally the instrument should be heard at an equal distance from either ear. If both sides are equally affected the hearing of the patient must be compared with that of a healthy person. In order to determine whether the deafness is neurotic or due to obstructive disease we test the sharpness of hearing through bone-conduction (Rinne's test). If the cause is middle-ear disease, impacted cerumen, or obstruction of the Eustachian tube, a vibrating tuning-fork placed upon the vertex will be heard much more intensely on the deaf side. In certain middle-ear diseases, however, as, for example, ankylosis of the bones, this does not hold true.

Hyperæsthesia of the Auditory Nerve. Very rarely in certain cases of facial paralysis, and not rarely in hysteria, there is abnormal acuteness of hearing (oxyacoia). In some individuals suffering from hemicrania or tic douloureux, and in meningitis, the hearing of certain sounds—for example, high musical notes and whistling, is accompanied by pain. Nervous patients often complain of subjective noises, buzzing, roaring, hissing, and singing—the so-called tinnitus aurium.

Paralysis of the Auditory Nerve. No case of absolute unilateral deafness due to a focal lesion in a hemisphere has as yet been observed. Deafness from disease of the auditory nucleus is very rare. That due to disease of the peripheral nerve is much more common. We may have a rheumatic auditory paralysis similar to that of the facial nerve, or the deafness may be due to pressure from a tumor or inflammatory exudate at the base of the brain, or disease of the mastoid process of the temporal bone. The localization of the lesion is often extremely difficult. The only positive point is that labyrinthine disease is apt to be accompanied by vertigo, while in disease of the nerve trunk vertigo is absent. Deafness due to occupation is worthy of mention. It is

not uncommon in blacksmiths, boiler-makers, locomotive engineers, and firemen. In some instances the patients can hear better during the noise incident to their work than when the surroundings are absolutely quiet.

Ménière's Disease. Aural Vertigo. We may define vertigo as a subjective feeling of motion referred by the patient either to his own body or to surrounding objects, with loss of equilibrium and without unconsciousness.

In this disease, first described by P. Ménière in 1861, there is paroxysmal vertigo, sometimes so sudden and intense as to throw the patient to the ground, tinnitus aurium, nausea, pallor, clammy sweat, and vomiting. The severity of the attacks varies greatly. There may be momentary unconsciousness. There is sometimes jerking of the eyeballs, nystagmus, or diplopia. The disease is paroxysmal in character, but slight vertigo and tinnitus are apt to persist between the attacks. Some deafness is present. The attacks may vary in frequency from several in a day to only one in several months.

Paralyzing Vertigo. Gerlier describes a remarkable form of paroxysmal vertigo accompanied by weakness, paresis in the extremities, drooping of the eyelids, marked lassitude and depression without unconsciousness. It occurs only in men and is epidemic in the Canton of Geneva.

The Glosso-pharyngeal Nerve. *The Ninth.* This nerve supplies the posterior third of the tongue with nerves of taste. It sends motor branches to the stylo-pharyngeus and the middle constrictor of the pharynx, and branches of common sensation to the upper part of the pharynx. To test the sense of taste the eyes should be closed, the tongue protruded, and small quantities of bitter, sweet, sour, and salty substances applied to various parts. The sensation should be perceived before the tongue is withdrawn.

We know but little about central diseases of this nerve. The situation of its cortical centre is unknown. A central paralysis of taste manifesting itself only on the posterior third of the tongue has never been observed. Peripheral loss of taste (*ageusia*) may be caused by affections of the mucous membrane, and is often met with in fevers and the coated tongue of dyspepsia. Perversion of taste (*parageusia*) is found in hysteria and insanity.

The Pneumogastric Nerve. *The Tenth.* This nerve supplies the pharynx, larynx, lungs, heart, œsophagus, and stomach. It may be compressed by tumors or inflammatory exudates. It has been tied in ligating the carotid and cut in removing tumors of the neck. Its nucleus may degenerate and the nerve may be the seat of neuritis.

Pharyngeal Branches. These, with branches from the glosso-pharyngeal, supply the muscles and mucosa of the pharynx. In paralysis of them, either from nuclear or peripheral disease, there is difficulty in swallowing, and the food does not pass into the œsophagus, but into the larynx and posterior nares. Spasm is always functional.

Laryngeal Branches. The superior laryngeal nerve supplies the mucosa above the vocal cords and the crico-thyroid muscle. The recurrent laryngeal supplies the mucosa below the cords and all the

muscles of the larynx except the crico-thyroid and the epiglottidean. All the motor branches arise from the spinal accessory.

Bilateral Abductor Paralysis. The posterior crico-arytenoids being involved, the glottis is not opened during inspiration; the cords are close together, so that there is stridor. Phonation is unimpaired. The affection occurs in tabes, bulbar paralysis, and hysteria.

Unilateral Abductor Paralysis. Pressure from an aneurism is the most common cause. The cord on the affected side does not move on inspiration. The voice is hoarse, and rarely there is dyspnœa.

Adductor Paralysis. There is palsy of the lateral crico-arytenoid and arytenoid muscles. The cords cannot be brought together when phonation is attempted.

The following table from Gowers shows the different conditions well:

Symptoms.	Signs.	Lesion.
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low-pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy.
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line not moving during inspiration, the other normal.	Unilateral abductor palsy
No voice; perfect cough; no stridor or dyspnœa.	Cords normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

Laryngeal Spasm. The adductor muscles are affected. Under the name of laryngismus stridulus it is frequently met with in children. It is the cause of the laryngeal crisis in tabes. There is no cough nor hoarseness, but respiration ceases, the face becomes congested, there is a struggle for breath, and as the spasm relaxes there is a deep inspiration, with a loud crowing sound.

Cardiac Branches. The heart's action is controlled by this nerve. Irritation may produce slowing of the pulse. In the case of Czermak it was possible to stop the heart for a few beats by pressing on a small tumor in the neck. If the nerve be palsied there may be increase in the frequency of the pulse. Normally the heart acts without consciousness participating. All sensations of cardiac palpitation and pain are conveyed to the brain through this nerve.

Pulmonary Branches. The motor branches supply the bronchial muscles. Asthma may be a neurosis of this nerve.

Gastric and Œsophageal Branches. These supply all the motor fibres to the stomach and œsophagus. Vomiting is caused either by direct irritation of them or reflexly, as in meningitis. Gastralgia is due either to a cramp of the stomach or direct irritation of the peripheral ends.

The Spinal Accessory Nerve. *The Eleventh.* The internal branch joins the pneumogastric nerve and passes to the laryngeal muscles. The external branch supplies the sterno-mastoid and, in part, the trapezius muscles. Disease of it causes complete palsy of the former and partial palsy of the latter muscle. The head is rotated with difficulty to the sound side. The shoulder droops a little, and the angle of the scapula is rotated inward by the rhomboids and the levator anguli scapulæ. There is difficulty in raising the arm, because the scapula cannot be fixed. There is no torticollis.

Spinal accessory spasm (torticollis, wry-neck) may be congenital. The sterno-mastoid is atrophied, hard, and shortened. In almost all cases there is facial asymmetry, the palsied side being the smaller.

Spasmodic torticollis may be tonic or clonic. In the former the occiput is drawn toward the shoulder of the affected side, the chin is raised, and the face turned toward the sound side. In the latter the head is drawn forcibly every few minutes in the same direction. In some cases there is severe pain. The sterno-mastoid may be affected alone or with the trapezius, and quite frequently with the splenius, the scalenus and platysma myoides, the rectus, and the obliquus. In time the muscles become markedly hypertrophied. If the muscles of both sides are affected the head is drawn backward. This disease is usually considered as a functional neurosis, but it is probable that some cases are due to disease of the cortical centres.

The Hypoglossal Nerve. *The Twelfth.* This is the motor nerve of the tongue and, to a great degree, of the muscles attached to the hyoid bone. Palsy of the tongue may be due to supra-nuclear, nuclear, or infra-nuclear disease. In the first place there is hemiplegia, no wasting, nor change in the electrical reactions. The tongue is protruded toward the affected side. In the second the lesion is apt to be bilateral, in which case the tongue lies motionless on the floor of the mouth, and speech and deglutition are much interfered with. There is atrophy and reaction of degeneration. The condition is likely to be part of a general bulbar palsy. In the third only one nerve is affected, and wasting and reaction of degeneration are present.

Rarely there occurs a *clonic spasm*, in which the tongue is thrust in and out many times in a minute.

The Spinal Nerves. Neuritis.

Traumatic injury, injury resulting from exposure to cold, and the injury that is inflicted by poisons, as that of rheumatism, gout, syphilis, lead, alcohol, and the toxins of specific diseases, as smallpox, typhoid fever, diphtheria, and other affections, may set up inflammation of the nerves. Neuritis may also be caused by direct action of bacteria, infection of the body having taken place in other situations.

Symptoms. The inflammation may be very intense and involve a large number of nerves. One only may be affected. The process may be moderate in degree. The symptoms, therefore, vary. The local symptoms are referred to the affected nerve and to the tissues in the area

of its distribution. *Pain* is the most common. It is of a boring or burning character, and is worse at night. It is increased by movement, by pressure, and by position. It may radiate to distant parts. The pain may not be confined to the nerve alone, but extend to the structures supplied by the nerve. The bone may be tender on pressure. The nerve, if accessible, is found on palpation to be swollen and extremely tender. *Vasomotor symptoms* are observed. The skin over the affected nerve is red, and may be œdematous. Eruptions may occur in the course of the nerve. In chronic cases trophic changes in the skin take place. Changes are observed in the nails (see page 133). Numbness, tingling, and other *paræsthesiæ* are complained of. The area of nerve supply may be *hyperæsthetic;* sometimes sensation is lost in small areas. *Wasting* of the muscles occurs; paresis, if not paralysis, is seen.

The *general symptoms* are moderate, although fever may be high and at its onset preceded by a chill.

In *chronic neuritis,* pain is the most common symptom. Trophic changes are more liable to occur. A wasting of the muscles ensues. Reactions of degeneration are determined by electricity.

Diagnosis. Neuritis must be distinguished from *neuralgia.* In the latter the pain is intermittent, the nerve trunks are not tender, while points of pain are more prominent in local situations. The diminution of sensation is an indication of the occurrence of neuritis.

Inflammation of Special Nerves. The following nerves are frequently the seat of neuritis: 1. The *phrenic nerve*—rarely (see Dyspnœa). 2. Other nerves of the brachial plexus. One nerve or the entire plexus may be affected. . The symptoms are the symptoms of neuritis and the symptoms that occur in paralysis of muscles supplied by the special nerve affected. *a.* The *posterior thoracic nerve.* The serratus muscle is affected. The paralysis is recognized by recession of the posterior edge of the scapula from the thorax when the arm is put forward. *b.* The *supra-scapular* nerve. There is paralysis of the supra- and infra-spinatus muscle. Ability to rotate the humerus outward is lost. *c.* The *circumflex nerve.* The deltoid muscle is paralyzed. Power of raising the arm is lost. When the muscle atrophies, the shape of the shoulder is changed. It must not be confounded with ankylosis of the joint. In ankylosis the scapula moves when the arm is moved. In paralysis it remains fixed. *d.* The *musculo-cutaneous nerve.* The flexors of the elbow are affected. The biceps and brachialis muscles are paralyzed. *e.* The *musculo-spiral nerve.* The triceps, the muscles in the back of the forearm, and the extensors of the wrist and fingers are affected. The symptoms are those of wrist-drop when the extensors are affected. The triceps muscle often escapes because the nerve is affected below the point from which the branches which supply this muscle pass off. The power of supination is also lost. The muscles waste; the extreme flexion causes prominences about the wrist and hand. There is marked degenerative reaction. Sensation is variable; it may be lost. The affection is usually unilateral, whereas in lead-poisoning it is bilateral. *f.* The *median nerve.* The flexors of the fingers, the abductors and flexors of

the thumb, the pronators and the radio-flexor of the wrist are affected. Pronation is markedly interfered with; flexion of the second phalanges on the first is lost. The wrist is flexed toward the ulnar side. *g*. The *ulnar nerve*. The ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, the muscles of the little finger, the interossei, and adductors of the thumb are affected. Its sensory areas are also affected.

3. The *nerves of the lower limb*. The symptoms are limited to the individual nerve trunks and their respective functional areas. The nerve of the leg most frequently affected is the sciatic—a neuritis of common occurrence. The onset is sudden, the pain is extreme; there is flexion of the leg in order to prevent tension of the nerve. The pain is intense in the course of the nerve trunk from a point above the hip-joint to the back of the foot. Tenderness on pressure is extreme. Abnormal sensations are very common. The muscles, especially the calf muscles, become flabby, and sometimes waste.

It must not be forgotten that in paralysis from neuritis in the nerves both of the arms and the legs, spinal cord lesions are closely simulated. In the arms, particularly, muscular palsy, wasting, and anæsthesia are often of spinal origin. The unilateral seat of the disease, and the local symptoms of the neuritis aid in the diagnosis. Neuritis must be distinguished from writer's cramp and other occupation neuroses.

Multiple Neuritis.

Multiple neuritis is a disease in which a number of nerves become inflamed simultaneously or successively. The nerves most frequently affected are those of the arms and legs, particularly the musculo-spiral and the anterior tibial; these become the seat of pain, swelling, and tenderness, and the extensor and flexor muscles supplied by them paralyzed, producing wrist- and foot-drop. Excluding diphtheria, leprosy, and the Japanese disease known as kakke-kakke, the most common causes are chronic alcoholism, cold, and exposure. It is most common in middle life, and females are said to be more frequent victims than males. It may be acute or subacute in its onset; when acute there may be marked fever with rigors. The initial symptoms are usually tingling, numbness, and dull pains in the limbs; the pains increase in severity and become shooting and burning, as in simple neuritis. The muscles of the limbs are tender to pressure, and the nerve trunks themselves highly sensitive and sometimes perceptibly swollen. The affected muscles lose power, waste, and show degenerative reactions. The skin is at first hyperæsthetic, but subsequently often becomes anæsthetic to touch while hyperæsthetic to pain. The deep reflexes are lost, and vasomotor and trophic changes in the skin and its appendages and in the joints sometimes occur.

As a rule, the disease increases in severity for a few weeks and then slowly improves, but palsy may persist for months; it usually improves first in the legs and last in the arms. Death may occur from extension of the palsy to the respiratory muscles, but is rare.

Diseases of the Spinal Cord and its Membranes. Meningitis.

Inflammation of the dura mater (external meningitis) may be *acute* or *chronic*. The acute form is characterized by local pain in the back, increased by motion and pressure; by rigidity of the muscles; by radiating pains in the trunk or limbs, due to pressure upon the nerve roots, and hence called root-pains; and by hyperæsthesia, perhaps followed by anæsthesia, of the skin. In proportion to the extent of the irritation of the nerve roots, numbness, tingling, formication, twitching, and spasm accompany the pain; and if compression be sufficient, paralysis of motion results, with or without loss or perversion of sensation. The paralyzed muscles are flaccid, and reflex action is abolished. Urine and fæces may escape involuntarily from paralysis of the respective sphincters, and if the patient survive long enough bedsores may form. The disease is febrile, and, should pus form, the fever becomes high and is accompanied by chills and sweating.

As the disease is almost always consecutive to disease of the vertebræ, such as caries or traumatism, or to extension of suppuration from adjacent tissues, as in bedsores, the early symptoms are liable to be overlooked in the primary affection.

Gowers says that in case of apparently primary meningitis a careful watch should be kept on the tissues of the back; any sign of deep œdema in the muscles beside the vertebral column, in such a case, is probable evidence of commencing purulent inflammation extending from within, and the development of acute local inflammation in either the pleura, posterior mediastinum, back of the abdomen, or behind the pharynx, has the same significance.

The *chronic* form is characterized by the same symptoms of local vertebral pain, radiating root-pains with disturbances of sensation, together with symptoms of pressure on the cord. The pain is less acute, and the course of the disease much longer.

Inflammation of the pia and arachnoid (*internal*, or *lepto-meningitis*) may be *acute* or *chronic*.

The *acute* form is characterized at its onset by chill, fever, and local vertebral pain, which are rarely preceded by other symptoms. The pain rapidly becomes intense, is aggravated by motion, and is felt over a considerable portion of the spine, but is often worse at some part. In addition to the local pain, there are root-pains of great intensity, shooting into the trunk and extremities. There is a tendency to muscular spasm, showing itself first in stiffness of the muscles of the back, often causing rigidity and retraction of the head, and sometimes opisthotonos. Elsewhere the muscular spasm is exhibited in painful cramp of the abdominal muscles and muscles of the extremities. The latter become rigid, painful on pressure, and are liable to painful croup-like spasms on motion. The skin is hyperæsthetic, and reflex action, both of the skin and muscles, is increased. The bowels are constipated and the urine retained, from spasm of the sphincters. Spasm of the chest muscles sometimes causes intense dyspnœa. Swallowing, also, may be difficult. If the inflammation extend to the medulla, cerebral symptoms are superadded, such as delirium and coma. If the disease

progresses unfavorably the irritative symptoms give way to paresis and then to paralysis, accompanied by loss of sensation and reflex action. Recovery at this stage may occur, with gradual abatement of the pain and slow regaining of muscular power; or death may result from weakness and failure of respiratory power, or more slowly as the result of complications, such as bedsores and nephritis. The disease may also pass into a subacute or chronic form, loss of power gradually taking the place of the irritative symptoms, and atrophy and contractions appearing. The final result may be a chronic myelitis, or complete but very gradual recovery.

The disease is febrile, but the temperature may be only slightly above normal. The duration of the disease is from a few days to two or three weeks; but disturbances of motion and sensation may persist for months or even become permanent.

The disease may be traumatic in origin, or may arise from exposure to cold or to heat, including long-continued exposure to the sun. It may also be secondary to an external meningitis or to a cerebral meningitis, or be septic in origin.

Chronic meningitis differs widely in its symptoms from the acute form, particularly in the fact that spasm is almost wholly absent. There is local pain in the back which, as in acute meningitis, is increased by pressure and motion; but the pain is not so acute. The muscles are more rigid than normally, and there may be retraction of the head. Root-pains are severe and of a varied character. Hyperæsthesia to pain and touch may be marked. Muscular twitchings may occur, but they are not pronounced, and rarely amount to spasms. The parts affected by the radiating pains will of course depend upon the seat of the lesion.

After the lapse of weeks or months, loss of power occurs in the affected muscles. The radiating pain may continue or disappear. The paralytic phenomena are progressive, the muscles waste, reflex action is abolished finally, sensation is impaired, at least in the affected muscles. If the inflammation involves the lumbar enlargement reflex action is lost and atrophy of the legs occurs; whereas if it is above the lumbar enlargement, the reflexes, if lost temporarily, are regained, and wasting of the leg muscles does not occur.

Gowers says that in the trunk loss of reflex action with anæsthesia is of much diagnostic importance. There may also be some loss of coördination.

Cervical *hypertrophic pachymeningitis* (Charcot and Joffroy) closely simulates progressive muscular atrophy. Its earlier stage is characterized by pain in the back of the head, neck, shoulders, and arms, followed by wasting of groups of muscles of the arm and hand, leading to the deformity known as *main en griffe* (claw-hand), and to weakness and wasting of the muscles of the leg.

Chronic *syphilitic meningitis* is characterized by a tendency of the inflammation to localize itself to one part, and hence by unilateral radiating pains, anæsthesia, and paresis.

Meningitis has to be distinguished from muscular rheumatism, myelitis and tetanus. In *muscular rheumatism* of the back the pain is

local, and while increased by pressure and especially by motion, it is not accompanied by shooting pains in the trunk and limbs, nor by disturbances of sensation and motion; moreover, fever is moderate or absent.

In *myelitis* without accompanying meningitis local vertebral and root-pains are slight or absent entirely, and paralysis occurs early and is not accompanied by spasm.

In *tetanus* initial fever is absent, the jaw muscles are early affected with tonic spasm (trismus), and general muscular spasms are easily provoked by touch or motion.

The symptoms of chronic meningitis vary according to whether the dura (*pachymeningitis*) or the pia and arachnoid (*leptomeningitis*) are principally involved, and also according to the extent of irritation of the nerve roots and involvement of the cord. In leptomeningitis local pain in the back, stiffness of the muscles, and hyperæsthesia of the skin are more marked than in pachymeningitis. Nevertheless root pains are present, and paresis of the legs from involvement of the cord sometimes occurs early. On the other hand, in pachymeningitis local vertebral symptoms are subordinated to the root symptoms, and muscular atrophy may be marked.

In general, the symptoms dependent upon irritation or structural alteration of the nerve roots are the most important for diagnostic purposes, and when taken in connection with the vertebral symptoms and their mode of onset are generally sufficient to differentiate the disease from the affections with which it is liable to be confounded.

Meningeal Hemorrhage. This may be between the dura mater and the vertebræ (extra-meningeal), or within the dura (intra-meningeal); the former is more common. The symptoms resemble those of meningitis, but are more sudden and violent in their onset. Pain in the back is severest usually at a point corresponding to the seat of hemorrhage. As in meningitis, there are pains shooting into the limbs, with numbness, tingling or formication, muscular spasms, and paresis or paraplegia; when the hemorrhage is small there may be only paræsthesia and paresis of the extremities. If the hemorrhage be large there may be immediate paraplegia. As a rule paralysis does not become complete.

The hemorrhage may be due to traumatism, to severe convulsions, violent exertion, or rupture of an aneurism.

It needs to be distinguished from *hemorrhage into the cord* and from *meningitis*. In the former case vertebral pain is not so prominent a symptom as in meningeal hemorrhage, and is not usually so extensive. On the other hand paralysis is immediate, not gradual in onset, though it may be slight at first and then extend rapidly. Spasm is absent in hemorrhage into the cord, and recovery from the paralysis is more gradual. If hemorrhage involve both membranes and cord, of course the symptoms of both lesions will be seen together.

The absence of fever helps to distinguish hemorrhage into the cord from meningeal hemorrhage. Moreover, the symptoms in the latter disease are of a more gradual onset. But a meningitis may be set up by the hemorrhage, in which case its symptoms will follow those of the hemorrhage.

Hyperæmia. *Hyperœmia* of the spinal cord is indicated by a feeling of fulness, weight, or aching in the back, by paræsthesia of various kinds, and perhaps by some increase in the reflexes, a feeling of heaviness in the limbs, and some motor weakness. The symptoms are relieved when the patient lies prone. Active congestion must occur from excessive stimulation of the cord and motor nerves—as in convulsions, excessive muscular exercise, or over-frequent coitus—because such conditions have produced hemorrhage; but the symptoms merge into those of incipient inflammation so as practically to be inseparable from it.

Anæmia. *Anœmia* of the cord is difficult to distinguish with sufficient definiteness except in general anæmia, or anæmia from sudden hemorrhage. In these conditions there is paresis of muscles, which may result in complete paralysis from subsequent degeneration of nerve elements. Sensation is usually not disturbed.

Compression of the Spinal Cord.

Compression of the spinal cord is most frequently the result of caries or fracture of the spinal column; but it occurs also in morbid growths, aneurism, and other conditions.

Pain is a prominent symptom; it is neuralgic in character, and may be felt in the upper or lower extremity or in the trunk; other symptoms are hyperæsthesia of the skin, followed by anæsthesia in places, without cessation of the pain ("anæsthesia dolorosa"). There is more likely to be motor weakness and atrophy than spasm. The motor weakness is at first overshadowed by the shooting pains, and as a rule deepens gradually into paralysis. But if the compression gives rise to myelitis, paralysis occurs rapidly. The reflexes are exaggerated. Sensation may or may not be impaired below the level of the compression. The symptoms are very rarely unilateral, though frequently one limb is affected first and to a greater degree than the other.

The diagnosis is based upon the existence of irritation of the nerve roots and cord, and upon the detection of some compressing cause. When the vertebræ are diseased, there is considerable local tenderness as well as pain, which is decidedly increased by movement. Additional diagnostic points are slow development, increase of reflexes, invasion of one side before and to a greater degree than the other.

As to the cause of the compression, Gowers states that if the patient is in the first half of life, and inherits a tubercular tendency, caries is suggested. The absence of root symptoms is also in favor of caries, but their presence does not render caries less likely unless the pain is extremely severe and is greatly increased by movement.

Recovery will depend for the most part upon the persistency or increase of the compression. If it be removed, or even if it cease to increase, recovery is often complete.

Myelitis.

Myelitis, or inflammation of the spinal cord, may be acute or chronic. It may involve the entire thickness of the cord through a short segment (*transverse* myelitis); it may involve continuously a large section of

the cord (*diffuse* myelitis); or scattered areas may be affected (*disseminated* myelitis); or one small area may alone be the seat of inflammation (*focal* myelitis); when the gray matter is wholly or chiefly involved it is called *poliomyelitis.*

Acute Transverse Myelitis is characterized by the rapid development of paralytic symptoms, impairment or loss of sensation, a girdle sensation at the level of the lesion, either increase or loss of reflex action, more or less atrophy of the affected muscles, and paralysis of the sphincters. The onset of the disease may be preceded by fever, headache, delirium, or gastric derangement; by rheumatoid pains; by paræsthesia in the limbs; or it may be ushered in abruptly by a convulsion. Convulsions, however, are rare, except in children. Vertebral pain is rarely marked and may be absent entirely.

Retention of urine is a very significant and important early symptom. The paralytic phenomena begin by a feeling of weight and weariness in the limbs, possibly accompanied by numbness and tingling. If the patient be walking he will be obliged to sit down to rest, and attempting to rise, he may find it impossible. More frequently the paraplegia develops more gradually and becomes complete only after the lapse of some days. In other cases after paresis has existed for several days paralysis supervenes somewhat suddenly.

Paralysis of sensation may be complete or variously impaired. Sensibility to touch may be lost while pain is felt. A hyperæsthetic zone usually exists immediately above the lesion, and its seat is detected by passing a hot sponge down the spine. When opposite the zone the sense of warmth becomes one of pain. The girdle sensation is felt at the same level.

The condition of the reflexes and of the nutrition of the muscles depends largely upon the seat of the lesion. If the lumbar enlargement is involved in the inflammation the reflexes are abolished and atrophy speedily follows. If above the lumbar enlargement, the reflexes may be lost temporarily, but are subsequently regained and become exaggerated, while atrophy to any considerable degree does not occur.

The urine and fæces are at first retained, and subsequently are passed involuntarily. Trophic changes in the skin predispose to ulceration and bedsores. Severe cystitis is not uncommon. Fever is present during the progressive stage of the disease, but is usually slight—99° to 101°.

The initial lesion may be the only one, or the inflammation may tend upward or downward; or, again, after apparent convalescence, there may be a fresh outbreak. In cases that end in recovery sensation is regained in the course of a few weeks or months, and eventually motion also. Spastic paraplegia may be left as a result.

Acute myelitis may result in death or in recovery, and the latter may be complete or incomplete. Death may occur early from interference with respiration, or occur later from the involvement of the medulla by disseminated myelitis, or be secondary to disease of the kidneys or of other organs, particularly to exhaustion or septicæmia from bedsores; when recovery occurs the restoration to power is slow.

Disseminated Myelitis is characterized usually by the consecutive

development of symptoms pointing to lesion of the cord at different levels. It requires for its exact diagnosis ability to differentiate the symptoms produced by various focal lesions. Gowers states that the onset of this form is often subacute, and that constitutional symptoms are often absent.

"An inflammation which continues to extend after the first two or three days is certainly disseminated, and most subacute cases are of this variety, and so are those that are secondary to blood states. The distinction is important, because this form is far more grave than any other and more likely to cause death."

Central Myelitis is the name given to inflammation of the gray matter surrounding the central canal of the cord. It is characterized by violence of onset and by a rapidly fatal course. There are complete paraplegia and complete loss of sensation in the lower limbs; the sphincters are paralyzed and reflex action abolished. Moreover, the affected muscles atrophy with great rapidity. Fever is marked, and death usually occurs in a few days.

Acute myelitis may arise from traumatism, from hemorrhage, or be secondary to meningitis. It may also arise from cold, particularly from lying upon the back on damp ground; from over-stimulation of the cord by sexual excesses; in the course of or during convalescence from the infectious fevers; and under the influence of gout, syphilis, and alcoholism.

Chronic Myelitis. Chronic myelitis, called also *diffuse myelitis, diffuse sclerosis, chronic transverse myelitis,* presents symptoms differing from those of acute myelitis chiefly in their slow onset. Its essential characteristics are the impairment of motion and of sensation, paræsthesia and sometimes dull pains in the legs, a decided girdle sensation, exaggeration of the reflexes, and usually not much atrophy.

The patient finds that the legs are heavy, and that they become tired easily. He walks slowly, does not lift his feet clear of the ground, but is inclined to drag them. The muscles become rigid, and as they grow weaker the reflexes are exaggerated until a condition of spastic paraplegia is reached. Sometimes there is loss of coördinating power, but no true ataxia. Constipation and slowness and difficulty in micturition indicate the impairment in expulsive power of the rectum and bladder. Sensation is not lost to the same extent as motion. There is often a constant dull pain in the back, and the affected limbs may be the seat of tingling, numbness, and formication. There is usually well-marked girdle sensation.

The disease may be widely scattered, and hence almost every symptom of spinal involvement may be met with.

If the gray matter is involved (*chronic poliomyelitis*) there are atrophy, anæsthesia, and paralysis. These develop with greater or less rapidity, sometimes involving the legs first and then the arms, and sometimes the arms first and then descending.

Chronic myelitis usually runs a very chronic course. It may progress steadily and uniformly, or at times grow worse rapidly; but at any period the disease may be arrested and become stationary. Its duration is therefore extremely indefinite, varying from one to twenty

years. Spitzka states that the average duration is from six to fifteen years.

Chronic myelitis is differentiated from *hysterical paraplegia* by the presence of degenerative reaction in the muscles, by the fact that incontinence of urine is more common in myelitis, while in hysterical paraplegia retention is the rule; by the absence of pupillary phenomena in the latter; and by the fact that anæsthesia, if present in hysteria, is less likely to correspond with the distribution of the motor paralysis. Moreover, the hysterical patient can overcome the paraplegia to a considerable degree by a strong effort of the will.

From *compression* of the cord it is distinguished by absence of any obvious cause of pressure, such as injury or caries of the vertebræ, and absence of root-pains, which would indicate that the process had begun outside the cord.

From *tumor of the cord* it is distinguished by the comparative absence of root-pains. Both may involve one-half of the cord more than the other, but myelitis is more likely than tumor to present absolutely unilateral symptoms.

From *primary lateral sclerosis* (spastic paraplegia) it is distinguished by the existence of both motor and sensory impairment, whereas in spastic paraplegia the symptoms are entirely motor.

From *progressive muscular atrophy* it is distinguished by the atrophies of myelitis being irregularly distributed, while those of progressive muscular atrophy are symmetrical. Moreover, in the former there are other cord symptoms and sensory disturbances.

Pachymeningitis is distinguished principally by greater pain and by a more pronounced and extensive anæsthesia. Gowers says that if there are similar symptoms in both arms and legs, myelitis is far more probable than pachymeningitis, since the chronic inflammation of the membranes is less extensive than that of the cord.

Anterior Poliomyelitis. This disease is also called atrophic spinal paralysis, and infantile spinal paralysis, etc. Children up to the fifth year are most frequently attacked, and invasion is more common in summer than in winter. Its essential characteristics are suddenness of onset with complete paralysis, which speedily abates to a certain extent, leaving certain muscles or groups of muscles permanently paralyzed; these waste rapidly and progressively, and lose their electrical contractility. Sensation is undisturbed, the sphincters remain unaffected, trophic disturbances of the skin are absent, and the intellect is not involved. The affected limbs are contractured.

The onset of the disease may be marked by fever, which is usually moderate, by convulsions or delirium, by rheumatoid pains; or it may appear without warning of any kind, either during the day, or be found in the morning after a quiet night. Fever, when it occurs, rarely precedes the paralysis more than a day or two. Sometimes the disease develops during the course of, or during convalescence from, one of the specific fevers. The extent of the paralysis varies; it may involve only one limb, or all four limbs and the trunk. If the child has been ill, or if the early symptoms have compelled the child to go to bed, paralysis

may be detected first when the child gets up, previous disability being attributed to general weakness or lack of energy.

The paralysis attains its greatest extent rapidly, often in a few hours; remains unchanged for from two to six weeks, and then begins to abate in the inverse order in which it began. That is to say, if the arm was first affected and the leg last, the paralysis in the leg will begin to improve first. This order of improvement is characteristic, and led Barlow to call the disease "regressive paralysis." All the affected muscles do not recover. Those which remain permanently paralyzed waste rapidly and display degenerative reaction.

The superficial reflexes are lost, but there is no loss of sensation, although paræsthesia may be felt. The bones may cease to grow in the affected limb, which therefore becomes shortened relatively to its fellow. Contractures are a late result.

Acute Ascending Paralysis. Acute ascending, or Landry's paralysis, is characterized by a rapid and progessive paralysis, beginning usually in the feet and extending upward, involving the muscles of the trunk, chest, arms, and neck; swallowing and speech may be abolished. Sensation is practically unaffected, though there may be paræsthesia and hyperæsthesia of the skin. Reflex action may or may not be regained.

The muscles are toneless and are neither atrophied nor changed in their electrical reactions. The disease is afebrile. Enlargement of the spleen has been noted in several cases.

The course of the disease is usually rapidly fatal, most of the patients dying within a week; but it may last several weeks, and recovery is not impossible.

The cause of the disease is unknown, and no lesions have been found post-mortem.

Diver's Paralysis. Diver's paralysis is generally a paraplegia; it comes on in persons who have remained at a considerable depth below the surface for at least an hour. It is more apt to occur after the diver has returned to the air than when he is in the water. It usually comes on very rapidly, sensation as well as motion being lost, and the lower half of the body feeling numb and foreign to the patient. Recovery generally occurs in from three to ten days, but it may be much slower than this, and may in rare cases be permanent. Death occurs occasionally.

Hemorrhage into the Spinal Cord.

Hemorrhage into the spinal cord, or hæmatomyelia, is extremely rare clinically. The symptoms produced are those already described as occurring in acute transverse myelitis, from which the essential point of difference is the great suddenness of onset. It may arise from injury, over-exertion, and sexual excess.

The prognosis depends upon the size of the hemorrhage and upon its seat.

It is better in proportion to the rapidity with which sensation is regained. Myelitis may, however, be a secondary result.

Degenerations of the Spinal Cord.

1. Locomotor ataxia.
2. Primary spastic paraplegia.
3. Ataxic paraplegia.
4. Chronic muscular atrophy.
5. Arthritic muscular atrophy.
6. Pseudo-hypertrophic muscular paralysis.
7. Thomsen's disease.
8. Tumors.
9. Syringomyelia.

Locomotor Ataxia. Locomotor ataxia, frequently also called tabes dorsalis, also posterior sclerosis, is a chronic degenerative disease of the spinal cord, involving the posterior columns and root fibres, and characterized by lightning pains, usually felt in the legs, by absence of knee-jerk, and by incoördination of movement without paralysis or muscular wasting.

The lightning pains and loss of knee-jerk precede the incoördination, which very rarely is absent.

Incoördination of movement is of gradual development. The patient usually first notices that at night he cannot walk without stumbling, though during the day he walks well enough. Or he may not have noticed any loss of coördination himself, but when examined by the physician it will be found that when asked to close his eyes and then walk, he staggers and would fall unless supported; and that he is unable to maintain his equilibrium when standing with the feet close together, unless the eyes are at the same time open. The reason for this is that the muscular sense and sense of position are deficient, and without the guidance of vision the patient cannot tell where he is. By degrees incoördination becomes manifest, even when the eyes are open. The gait becomes, in time, characteristic; the leg is thrown laterally and forward with a jerk, and then brought down suddenly and forcibly, the whole sole striking the ground. Finally, he may be unable to rise to his feet, as any attempt to rise, or contact of his feet with an object, produces spasmodic, pendulum-like motions. Incoördination may affect the arms also, but almost always after the legs have been affected. Ataxia may be developed on making attempts to write, or to button and unbutton the coat. The muscles retain their power, except in advanced cases, when there may be some weakness.

Disturbances in sensation are very marked and are very rarely absent. Darting pains in the legs, called from their suddenness and severity "lightning" pains, are characteristic. They are paroxysmal, and while usually felt in the legs, may shoot into the arms, head, or other parts. The pains are not always lightning in character, but may be ordinary neuralgic or rheumatoid pains. Painful girdle sensations may be felt in the trunk and limbs. Paræsthesiæ are frequently complained of and partial anæsthesia is common later in well-marked cases. The perception of sensation may be considerably retarded.

The cutaneous reflexes are usually lessened, but in the early stages may be greatly exaggerated. Loss of sexual power is the rule; it may

occur early in the disease, or be a sequel to abnormally increased passion. The deep reflexes, particularly the knee-jerk, are almost invariably absent in the affected territory.

The rectum and bladder are more often sluggish in action than paralyzed. The eye symptoms are optic atrophy, paralysis of the ocular muscles, and the Argyll-Robertson pupil, *i. e.*, a pupil which contracts to accommodation but not to light.

A great variety of vasomotor and trophic symptoms may be present, such as œdemas, local sweatings, skin eruptions, atrophies, and joint changes.

The name *crisis* is used in tabes to describe the paroxysmal derangements of the functions of various organs which occur in the disease. The most common are gastric crises, in which there is severe pain in the stomach followed by vomiting, which may or may not be attended by nausea. Any organ may be subject to corresponding crises; thus we have at times laryngeal, rectal, or vesical crises.

The course of the disease is extremely chronic. Gowers says it is exceedingly common for the first stage—in which there is no alteration in gait, but loss of knee-jerk, pain, often Argyll-Robertson pupil, and unsteadiness on standing with the feet together and the eyes shut—to last for from ten to twenty-five years. He does not think the disease shows a progressive tendency in more than half the cases in which it is recognized early and carefully treated.

There is no general rule in the matter of progress. Often one symptom improves, and another appears or is aggravated. The disease itself is not fatal. Death may result from complications involving the kidneys and heart, or from some other nervous disease. As the primary cause of locomotor ataxia is in most cases syphilis, any other tertiary or secondary manifestation of syphilis may be found to coexist.

Primary Spastic Paraplegia. Primary spastic paraplegia, or primary lateral sclerosis, is a chronic degenerative disease of the cord, probably involving the pyramidal tracts or their terminations in the gray matter. It is characterized by a gradually developed loss of motor power in the lower extremities, spasmodic contractions of the muscles, with exaggerated reflexes, absence of wasting, maintenance of sensation, involvement of the sphincters, and a very chronic course.

The combination of rigidity with spasm makes the gait peculiar. In fully developed cases the patient cannot easily bring the foot forward; it drags behind and the toe has a tendency to stick into the ground; and as clonus is easily excited, there may be spasmodic contractions after the foot touches the ground. Sensation is, as a rule, maintained undisturbed, but paræsthesiæ may be present.

The arms as well as the legs may be involved, or only one arm and the corresponding leg. The disease may also be congenital. This form is distinguished, according to Gowers, by the wide separation and irregular movement of the fingers on attempting to take hold of an object. From pseudo-hypertrophic paralysis it is distinguished by exaggeration of the patellar reflex, absence of wasting, and presence of clasp-knife rigidity. "The impairment of locomotion gradually lessens in birth-palsy, while it increases in pseudo-hypertrophic paralysis."

The prospect of arrest of the disease and improvement of the paralysis is better in the infantile form than in adults, but even in them it may occur. As a rule, however, arrest of the disease is as much as can be hoped for. It is not fatal in its tendency.

The disease is distinguished from *locomotor ataxia* by the exaggeration of reflexes instead of their abolition, and by the absence of eye symptoms, lightning pains, and painful crises. Other portions of the cord are at times involved and give rise to disturbances of sensation or to muscular atrophy. *Hysterical paralysis* is excluded by the presence of spasmodic rigidity, with excessive knee-jerk and ankle clonus.

Ataxic Paraplegia. Ataxic paraplegia, or lateral and posterior sclerosis, presents characteristics of both locomotor ataxia and paraplegia. The prominent symptoms are the very gradual development of motor weakness and of loss of coördinating power. The weakness is first noticed in walking, and the loss of coördination at night or when the eyes are closed. The flexor tendons are more affected than the extensors. The weakness and lack of coördination increase gradually until the gait becomes feeble and tottering and finally impossible without constant support. Up to this point the disease resembles locomotor ataxia, but it is distinguished from it by the absence of lightning pains and the preservation of the muscle reflexes—indeed, the patellar reflex is much exaggerated, and ankle clonus is usually present. There may be dull pains in the back and legs. The arms may or may not be involved. The muscles do not atrophy. Eye symptoms are usually, not always, absent. There is some loss of power over bladder and rectum, but it does not amount to paralysis, and not often to considerable paresis.

The sexual power is lost, but it may be regained for a time. The progress of the disease is toward a condition of spastic paraplegia, the gait in which has already been described, the loss of coördination becoming less marked as the paralysis increases. Cerebral symptoms, beyond loss of memory and occasionally defect in speech, are absent.

The disease runs a very chronic course, and is not fatal in itself. Death, as in other degeneration, results from complications, particularly kidney disease and bedsores. The disease is distinguished from *locomotor ataxia* by the presence of the patellar tendon reflex; from *spastic paraplegia* by the presence of incoördination; and from *chronic myelitis* by the absence of girdle sensation.

Hereditary Ataxia. Hereditary ataxia, Friedreich's disease, or hereditary ataxic paraplegia, is a special form of ataxia which differs in the following important particulars from the ordinary form. It is hereditary; it develops most frequently in childhood and at the age of puberty; it attacks males and females with about equal frequency; lightning pains are usually absent; and there is greater tendency for the disease to involve the arms and to affect speech.

The disease develops gradually. Incoördination, first of the legs and then of the arms, is the most obtrusive symptom. The muscle reflexes are abolished. Nystagmus is the most constant ocular symptom. The effect upon sensation is variable; sometimes it is impaired and at others it is entirely normal.

The duration of the disease is very chronic—from ten to thirty years. Gowers says the only guide to individual prognosis is the observed rate of progress.

Progressive Muscular Atrophy. Progressive muscular atrophy, wasting palsy, chronic poliomyelitis, or amyotrophic lateral sclerosis, is due to a combined degeneration of the multipolar cells in the gray matter of the anterior cornua, and of the pyramidal tracts.

The disease usually attacks an arm first, and either the hand or shoulder muscles; and next in frequency, a leg. Preceding any noticeable weakness of the affected member there is sometimes aching and an unaccustomed feeling of weariness after its use. Sometimes, however, wasting is the first thing that attracts attention, particularly if the hand is affected first, for here wasting of the interossei makes a characteristic appearance. The corresponding leg is not usually noticeably affected during the first six months. The atrophy is almost always steadily progressive, involving the muscles of the chest and neck, in addition to those of the legs and arms. Loss of power accompanies the atrophy. As a rule this loss is most marked in the arms, while the legs, before wasting becomes marked, are in the condition described under spastic paraplegia. The muscle bundles often exhibit fibrillary twitchings. The atrophied muscles give characteristic degenerative reactions.

Respiration is much embarrassed from involvement of the diaphragm and external respiratory muscles. The face generally escapes, but speech is involved from extension of the disease to the medulla, and glosso-labial paralysis is simulated.

Sensory symptoms rarely amount to more than dull pains, except when there is an associated meningitis. The sphincters are not usually involved, but sexual power is generally lost. In advanced cases the affected limbs, especially the upper extremity, are wasted so that they appear like skin stretched over the bones.

The average duration of the disease is said to be about three years, but the progress may be more or less rapid than this in individual cases. It rarely becomes arrested. Gowers says that wasting which has existed for six months will probably persist unchanged. The chief dangers to life are pulmonary complications and bulbar paralysis.

Pseudo-Hypertrophic Muscular Paralysis. Pseudo-hypertrophic muscular paralysis is a primary disease of the muscles, consisting of an overgrowth of connective tissue and subsequent atrophy of the muscle. The disease occurs almost always in childhood, sometimes being noticed as soon as the child begins to walk, and it may be con genital. The calf muscles are first involved, and hence the child is apt to be slow in learning to walk. The gastrocnemii are apparently much enlarged, though this enlargement may be concealed in a fat child. It stumbles and falls in attempting to run, and is unable to raise itself on tiptoe. The calf muscle is at first much harder than normal, and subsequently becomes softened through increase of lipomatous tissue. The legs may be recognized as weak for months or even years before characteristic changes are detected in the muscles; but usually the apparent hypertrophy can be noticed within a few

weeks or months after weakness has become manifest. Gradually other muscles become affected, the infra-spinatus most frequently. Gowers attaches great diagnostic importance to the coexistence of enlargement of the infra-spinatus and wasting of the latissimus and lower part of the pectoralis. As atrophy and accompanying weakness increase, change of position is accomplished with more and more difficulty; the feet are spread wide apart, and the gait is oscillating ("duck-like"). If prone upon the ground, the child raises himself first upon his hands and knees, then extends the knees and rests upon toes and hands, then places one hand upon a knee with the other remaining upon the ground, and then pushes himself upright from this position.

Contractions and deformities are a later stage, the most important of which are club-foot and curvature of the spine. Sensation and the functions of the bowel and bladder are unaffected. The course of the disease is progressive, but very slow. Gowers states that severe pulmonary disease generally ends life some time between twelve and twenty. Few patients reach the age of forty. The course of the disease is slower in girls than in boys.

Simple Idiopathic Muscular Atrophy. Simple idiopathic muscular atrophy differs from the pseudo-hypertrophic form in that it occurs in families, that it presents no apparent hypertrophy of muscles, and that the palsy involves the face, occurs at a later period, and affects females equally with males.

The disease develops very gradually and affects persons with an hereditary tendency to the disease. It occurs most frequently between the fifteenth and thirtieth year, but may appear in infancy and after middle life. Atrophy and loss of power go hand-in-hand, appearing first usually in the upper arms, legs, or face. Unlike progressive muscular atrophy the deltoids are not usually involved, the disease attacking the biceps, triceps, long supinator, and external muscles of respiration. Eventually both sides are affected, though the disease frequently begins on one side. The facial expression changes; the lower lip juts forward, the lips are held apart, and the labio-nasal furrow is obliterated, giving the face a dull and wondering expression.

In the lower limbs some of the thigh muscles are affected, but not the calf muscles. The diaphragm may also be involved. The muscles do not show degenerative reactions, fibrillary twitching is almost always absent, sensation is undisturbed, the functions of bladder and rectum remain unaffected, and trophic and vasomotor symptoms are absent.

There is no uniformity in the rate of progress of the disease; it may reach its extreme only at the end of a very long life, or in a decade. The disease has no direct tendency to kill. Gowers says that in the cases of most severe degree and rapid course the patient has usually died of phthisis.

Thomsen's Disease. Thomsen's disease is a rare congenital and hereditary affection, characterized by tonic spasm of the muscles when an attempt is made to put them in motion after a period of rest. If the attempt is persisted in, the spasm gradually lessens until free use of the parts can be obtained. The muscles do not waste and do not exhibit degenerative reaction.

Tumors of the Spinal Cord.

Tumors of the spinal cord may be syphilitic, cancerous, or tubercular. The prominent symptoms of tumor are pain and gradually developing paralysis. The character of the pain is that already described as root pain—darting and shooting; it is paroxysmal, very severe, sometimes agonizing, making life a burden. Local tenderness is not marked, and may be entirely absent. The pains often begin on one side and finally affect both sides. Paræsthesiæ and anæsthesiæ are also present. Muscular spasm is a further evidence of irritation of nerve roots; other symptoms are girdle sensations, paralysis, atrophy, and contractures. The paralysis, like the root pains, is often at first unilateral, but usually becomes bilateral in course of time. It begins first as a paresis and only gradually deepens into paralysis.

The superficial and deep reflexes are sometimes decidedly increased. There may be a difference in temperature upon the two sides.

The diagnosis of the seat of the tumor must be made from noting the level at which the cord functions are disturbed. Tumors of the cord, as distinguished from those of the membranes pressing upon or extending into the cord, are characterized by a relative prominence of paralysis and absence of root symptoms. It should be remembered that a secondary myelitis may be produced. If the lumbar enlargement is involved, or the cauda, reflex action is abolished; whereas if situated in the dorsal region or still higher up, reflex action is exaggerated; again, in tumors of the lumbar enlargement and cauda the legs atrophy. Tumors of the cervical region cause pain in the arms, and often atrophy, while the legs show excessive reflex action. There may also be interference with respiration.

Syringomyelia.

Syringomyelia is a chronic affection of the spinal cord, of congenital origin, characterized pathologically by the presence of cavities. During life it may give rise to no symptoms, and therefore be unsuspected. When symptoms do occur they consist of paresis, anæsthesia, and atrophy. Atrophy and anæsthesia affect the hands principally, and paresis the legs. The functions of the bladder and rectum are often deranged, and trophic changes in the skin may occur. The pressure sense may be lost in the arms. The duration of the disease from the time symptoms occur is from two to three years, and the result fatal.

Diseases of the Brain. Pachymeningitis.

Inflammation of the dura mater usually develops secondarily to disease of adjacent structures or to injury; its symptoms are to be picked out from those of the primary condition. The prominent symptoms are headache, fever, delirium, and perhaps convulsions. Fever is not a constant symptom. If pressure is exerted upon the cortical motor area there may be paralysis of the opposite limbs.

Leptomeningitis.

Inflammation of the pia and arachnoid may be acute or chronic; may be simple, tubercular, syphilitic, or epidemic (see Cerebro-spinal Fever); it may be confined to the cortex or base, or be general.

1. Acute Meningitis is characterized by the more or less sudden onset of headache, vomiting, delirium, and convulsions, accompanied by stiffness of certain muscles, especially of the muscles of the back of the neck, and later by paralysis and coma.

Headache is the most prominent symptom. It is most frequently frontal, but rarely may be general; is usually intense, and in paroxysms becomes maddening, causing the patient to shriek with the pain. It is aggravated by light and by sound or other vibration. Rarely, headache is absent; when this is the case, however, it is most frequently in the meningitis secondary to septic or blood diseases.

Vomiting is sudden and explosive, without antecedent or subsequent nausea or any local cause except the presence of food.

Delirium is usually active in type, and may be mild or almost maniacal. It is not often continuous, but is broken by lucid intervals. When coma appears it follows delirium.

Rigidity of the muscles of the back of the neck, in marked cases accompanied with retraction of the head, is an important symptom. Convulsions when they occur are general. They are more likely to occur in children than in adults. They may also be partial or unilateral, and so may paralyses. Cutaneous hyperæsthesia is not very uncommon. Neuritis of the optic nerve, according to Gowers, is a common symptom in meningitis of the base, but is rare when the inflammation is confined to the convexity. The most constant and important eye symptoms are strabismus and inequality of the pupils. The facial nerve may be affected, especially in meningitis of the base.

The range of *temperature* is far from uniform. Usually there is moderate fever from the start. Sometimes, especially in purulent cases, the fever is high and remains so until the patient's death. In fatal cases the temperature may either rise or fall on the approach of death, and in rare cases it may remain normal throughout. The pulse is not characteristic.

The disease lasts from one or two days to two or three weeks.

The symptoms vary somewhat according to the character of the inflammation. In simple meningitis the fever is more marked, optic neuritis is more common, and the duration is longer. Recovery may ensue.

Tubercular Meningitis is preceded by deterioration of the general health, emaciation, slight evening fever, peevishness, and sometimes distinct evidence of tubercle elsewhere, particularly in the lungs. Headache and apparently causeless vomiting are important symptoms; they may appear first at the onset of the disease, or may precede it by a short time. Other early symptoms are constipation, nightmare, irregular pulse, and cerebral hyperæsthesia, as the result of which light becomes painful, and slight sounds are disturbing. Loss of flesh continues, there is moderate fever, the abdomen becomes retracted,

the child loses strength, becomes apathetic, lying with its eyes partly open. It may be roused to temporary interest in its accustomed playthings, but soon turns from them in anger or disgust. Bright-red spots or streaks of hyperæmia may appear and disappear rapidly from the face. If the thumb-nail be pressed upon the skin and drawn across it, a red streak follows—the *tâche cérébrale.*

The child's sleep is disturbed by dreams, and it utters a peculiar piercing cry, the "hydrocephalic cry."

There may be some rigidity of the muscles of the back of the neck. Gowers lays particular stress upon the occurrence of aphasia. The eye symptoms are strabismus, irregularity of pupils, and optic neuritis.

Delirium and convulsions may occur early, but usually not until the second week. Local convulsions and corresponding palsies are common, but the palsy may be transient. Death may occur in convulsion, or more commonly in coma.

The temperature range is not constant, and often fluctuates considerably within short intervals. The pulse is often frequent at first, then becomes slow and irregular, and finally very frequent. The respiration is irregular and cerebral in type toward the close of the disease.

The duration of the disease is usually from one to three weeks, but it may be prolonged to twice that time. The prognosis is not necessarily fatal, but most patients die. The prognosis is graver when convulsions or coma appear early, and is better the longer coma is deferred.

Meningitis is to be distinguished from *general febrile diseases* with cerebral symptoms. Headache is common in the latter, but it is rarely so intense as in meningitis, unless there be at the same time high fever. Delirium is also common in both, but it succeeds the headache in febrile diseases, whereas in meningitis both symptoms persist together. Convulsions may occur at the onset of the exanthemata and pneumonia, but in meningitis they are a later symptom. Eye symptoms are absent in general febrile diseases. The best safeguard against a mistake in diagnosis is to examine every organ carefully before concluding that the mischief is in the brain membranes; particularly the lungs should be examined for a pneumonia, and the spleen and bowels for signs of typhoid fever. Tubercular meningitis is generally secondary, but it is not often possible to detect the primary focus.

Tubercular meningitis is distinguished from the *simple form* by the occurrence of premonitory symptoms of failing health in a child disposed by heredity to tuberculosis, or affected by an antecedent tuberculosis of bone, gland, or lung. It is further distinguished by the absence of other cause of meningitis, particularly traumatism, suppurative disease of the middle ear, infectious disease, such as erysipelas, or septicæmia. Moreover, the individual symptoms are important: tubercular meningitis is generally basilar; hence apparently causeless vomiting, strabismus, irregularity of pupils, and optic neuritis are very significant symptoms. The pulse in tubercular meningitis is often at first frequent, then becomes slow—40 to 60—and irregular and intermittent in the first stage, subsequently becoming frequent and irregular, and finally very frequent, but regular. The respiration is irregular and sighing and may be Cheyne-Stokes. The temperature also has a lower range and is

more fluctuating than in simple meningitis. Gowers says that tubercular meningitis is one of the most common causes of aphasia in children, and that it is sometimes an early symptom. Aphasia, however, is difficult to detect in children of the age at which tubercular meningitis is most common. On the whole, the disease is more liable to be suspected when it does not exist than to be overlooked when present.

In adults tubercular meningitis is rare and is always secondary, generally to disease of the lungs. It is, therefore, a late manifestation of the disease, except in the cases in which there is a general miliary tuberculosis.

Chronic Meningitis. Chronic meningitis is usually alcoholic or syphilitic, but it may be the result of sunstroke. The alcoholic form involves principally the convexity, and is characterized by headache, some loss of intellectual power, perhaps irritability of temper; there may also be occasional delirium and some optic neuritis.

In the *syphilitic* form the meningitis is more likely to be local, and usually extends from the seat of a gumma. The symptoms, therefore, are apt to be focal. Gowers says that it is highly probable that focal inflammation in adults is always syphilitic in nature, the traumatic form of course excepted.

The *purulent* form of acute meningitis most frequently affects the membranes of the convexity. It is characterized by high fever, with or without rigors, intense headache, vomiting, motor symptoms, possibly amounting to convulsions, and coma.

It may arise from mastoid disease, from injury, or be part of a general septic process. Its course is usually rapidly fatal.

Diagnosis. Meningitis is simulated by *brain tumor*. Loss of motor power in limbs indicates tumor rather than meningitis. Gowers says that if after the first two weeks from the commencement an optic neuritis continues to increase and the patient does not become comatose, the diagnosis of tumor is almost certain.

The most important symptom in differential diagnosis of meningitis from hysteria is increased temperature. When strabismus is present in hysteria it is convergent, never divergent. Gowers asserts that divergent strabismus or irregularity of pupil is certain evidence of organic disease, and as much so if it is transient as if it is permanent. In hysteria, also, there may be retention of urine, but never incontinence.

Cerebral Anæmia.

Cerebral anæmia may be a part of the general anæmia which characterizes chlorosis, leucocythæmia, and many other affections; or it may result from hemorrhage or other exhausting discharge. In other cases it is local, resulting from a deficient supply of blood to the brain. Such anæmia occurs in arterio-sclerosis, in aortic valvular diseases, and in aneurism of the aorta and its cervical branches.

If anæmia is suddenly developed, as from hemorrhage or a sudden assumption of the erect posture by a person with feeble circulation, the phenomena are those of faintness, ringing in the ears, dizziness, partial or complete blindness, general muscular relaxation, nausea, frequent,

feeble pulse, and shallow sighing respiration. The skin may become cold and be bathed in perspiration. The symptoms are aggravated by the erect posture.

When anæmia develops gradually the symptoms are less intense. Intellection is performed with slowness and difficulty, slight effort causes weariness and headache, and the patient is drowsy. Sight and hearing may be defective, and muscæ volitantes and tinnitus are common causes of complaint. There is usually some muscular weakness.

The diagnosis is not difficult.

Hyperæmia of the Brain.

Hyperæmia of the brain may be *active* or *passive*.

The diagnosis of *active congestion* is more liable to be suspected when it is not present than overlooked when present. The most trustworthy symptoms, according to Gowers, are the paroxysmal recurrence of headache, delirium, and sometimes fever, preceded by throbbing of the vessels and reddening of the face. The probability of active congestion is increased if the symptoms are relieved by nose-bleed or venesection.

The diagnosis of *passive congestion* is based upon signs of a plethoric habit, such as florid complexion, turgid vessels, associated with a dull, more or less persistent headache, which is aggravated by stooping, by coughing, constipation, or recumbent posture. Other symptoms are flashes of light before the eyes, slight dizziness, sluggish intellect with drowsiness, and some hyperæsthesia of the extremities. Slight convulsions sometimes occur. Passive congestion occurs in conditions which retard the escape of blood from the brain.

Cerebral Hemorrhage. (Apoplexy.)

Cerebral hemorrhage—that is to say, hemorrhage into the brain substance—is caused, apart from traumatism, by the rupture of a bloodvessel the walls of which have been weakened by disease and have become the seat of minute, or miliary, aneurisms.

The liability to it increases very markedly after the fortieth year. The symptoms differ considerably according to the extent of the hemorrhage and its seat; but the most frequent and prominent are sudden onset with loss of consciousness, convulsions, and coma, and, if recovery result, hemiplegia on the side opposite to the lesion.

Premonitory symptoms are present in a few cases. These may be those of cerebral congestion (*q. v.*) or consist of vertigo, change of temper, or vomiting. In some cases an unusual sense of well-being has preceded an attack. It is probable that these symptoms are really due to minute hemorrhages. The onset may be very abrupt, the patient falling unconscious as though struck upon the head. More frequently the loss of consciousness, while sudden, is preceded by headache, giddiness, faintness, nausea, or difficulty in articulation. If the seizure has occurred after a hearty meal the patient usually vomits freely and then becomes unconscious, with conjugate deviation of the pupils, the face

drawn to one side, the cheeks flapping with stertorous respiration, the lips covered with froth, and the arms and legs upon the affected side alternately convulsed and rigid, and relaxed.

If the attack comes on when the patient is standing, a weakness in one leg may cause him to fall or sit down, unconsciousness soon developing.

The degree in which consciousness is affected varies with the severity of the case. Usually it is completely lost, but it may be soon regained. Convulsions are most frequent when the hemorrhage is cortical. The pulse is usually slow and full, but it may be small, hard, and frequent. The respiration is stertorous, and may be Cheyne-Stokes. When convulsions are present they usually begin by twitching of the eyelids and eyebrows, rotation of the head and eyes by successive small movements to one side, usually the side of the brain lesion, and then the convulsion extends to the arm and leg and may become general. If consciousness is not completely lost the hemiplegia becomes very conspicuous; or if the seizure has occurred during sleep the patient may himself first become aware of it by the existence of hemiplegia when he attempts to get out of bed. When unconsciousness is profound (coma), urine and fæces may be passed unconsciously. In some cases there is an apparently mild seizure with rapid return of consciousness and power, except, perhaps, of speech, but in a few days the symptoms become worse and the patient dies comatose. The name *ingravescent apoplexy* has been applied to such cases.

If consciousness is regained and the patient recover, the symptoms are then those of palsy. This is most complete at first. It may be recovered from entirely, but usually recovery is only partial. Its extent and distribution depend upon the seat of the lesion. It is almost always unilateral.

The *seat* of the hemorrhage can be judged with tolerable accuracy. The most common seat is in the neighborhood of the corpus striatum and internal capsule, hence the frequency of hemiplegia. Cortical hemorrhage is rare. It is characterized by convulsions, which are local, and the resulting palsy may affect only a leg or an arm. A large hemorrhage into the *pons* causes deep coma, general paralysis, convulsions which are usually general, but sometimes involving only the legs. The pupils are contracted, there may be general anæsthesia, vomiting is common, and often high temperature. Death often occurs early. Hemorrhage into the *optic thalamus* causes a decided rise in temperature, but the palsy is slight.

Hemorrhage into the *medulla* causes death speedily without the occurrence of convulsions but with high temperature.

Hemorrhage into the *cerebellum* may or may not cause paralysis, and when it does the paralysis may be on the same side or on the side opposite to the lesion. It is attended by loss of consciousness and repeated vomiting, but vision is not affected.

Hemorrhage into the *ventricles* is marked by profound loss of consciousness, with conjugate deviation of the head and eyes. There may be temporary improvement, followed by complete coma with or without convulsions.

Meningeal hemorrhage is usually of traumatic origin. If the blood is poured out suddenly the symptoms are those of severe apoplexy, with rapid development of coma. If the escape of blood is more gradual there is often a period during which the patient is able to walk about; drowsiness then comes on, and deepens into coma. Less commonly there are convulsions, and sometimes delirium.

Diagnosis. The *coma* of *apoplexy* is distinguished from that of *alcoholism* by the presence of a drawn face and of more profound unconsciousness. Frequently the alcoholic can be roused sufficiently to grunt his disapproval or to turn over. The fumes of ammonia are said to rouse him. The temperature in alcoholic coma is depressed. The absence of convulsions is in favor of alcoholism. The respiration in the latter is quieter, and is not attended by frothing at the mouth or flapping of the cheeks. Mistakes are most likely to occur when no history of the patient's previous condition or of the mode of onset of the coma can be obtained. The odor of alcohol upon the breath is of value if the patient is known to be intemperate and if no one has administered alcohol subsequently to the coma. Incontinence of urine or of fæces is against alcoholism, and so is a bitten tongue.

Apoplexy is distinguished from *uræmia* by its sudden onset and comparative or complete absence of premonitory symptoms. In uræmia the patient has generally suffered from headache and morning nausea, which may be called by him "bilious attacks." The pulse is often of markedly high tension, and the second aortic sound is accentuated. In other cases there will be found œdema of the eyelids, a pale, waxy, bloated face, sometimes dropsy, and failure of vision. Marked drowsiness often immediately precedes an attack, and it is frequently accompanied by cramps and twitching of the muscles. The coma of uræmia is accompanied by stertorous respiration and frothing at the mouth; but the cheeks do not flap during respiration, and the face is not drawn as in apoplexy. The convulsions of apoplexy are more apt to be unilateral than those of uræmia, which are epileptic in type. They are often accompanied in apoplexy by conjugate deviation of head and eyes, but not in uræmia. Moreover, in apoplexy the skin is moist and warm, whereas in uræmia it is cool and dry and harsh. The temperature in apoplexy may be elevated at first, and then depressed; or it may continue to rise. In uræmia it is depressed. The condition of the urine is important in diagnosis, but it is not an infallible guide. A scanty, reddish, opaque urine, containing a large amount of blood and albumin, certainly points to uræmia; but apoplexy often occurs in a person of unsound kidneys, and its onset is frequently attended with the appearance of considerable albumin and of casts in the urine.

Cerebral hemorrhage is to be distinguished from *softening*, the result of *embolism*, by the age of the patient, the presence or absence of a cause of embolism, such as valvular heart disease and syphilis, and the intensity of the symptoms. Embolism is more frequent in those under forty; cerebral hemorrhage in those over forty. Intensity of apoplectic symptoms and persistent palsy are in favor of hemorrhage. When, however, the patient is past middle life the probability of softening does not diminish, and the diagnosis from hemorrhage must be made

by the symptoms and the condition of the patient. A high-tension pulse, hypertrophy of the left ventricle, and atheroma of the arteries of the limbs are in favor of hemorrhage; on the other hand, a weak heart and feeble pulse favor softening. If the attack comes on after much excitement or strong muscular effort, it is in favor of hemorrhage.

Premonitory symptoms, such as singing in the ears or paræsthesia of one side are in favor of softening. Profound coma and violent convulsions are probably from hemorrhage.

Cerebral thrombosis is characterized by more gradual onset, shorter duration of paralysis, and other symptoms, and by more complete recovery.

Thrombosis of the Superior Longitudinal Sinus.

This occurs most frequently in children; it may arise spontaneously in the course of acute diseases producing great prostration, especially entero-colitis. It results also, and more frequently, from inflammatory disease of the brain membranes or bone adjacent to the sinus, and extension of the inflammation to the walls of the sinus. The symptoms are the gradual development of coma with convulsions, which may be general or unilateral. Headache, stabismus, and more or less rigidity of the limbs are common. Adults are more likely to be affected with delirium than with convulsions. Epistaxis may occur, and sometimes there is œdema with distended veins upon the scalp and forehead.

The result is fatal in nearly all cases, but recovery is possible in spontaneous thrombosis.

Infantile Hemiplegia.

Infantile hemiplegia is an acute cerebral palsy occurring during the first five years of life; it is either primary or secondary to acute diseases, particularly scarlet fever and measles. The onset of the disease may be marked by vomiting and convulsions, by drowsiness or coma, or the child may wake up in the morning with well-marked hemiplegia. In other cases a series of convulsions precedes the appearance of the palsy, and in still other cases the onset is marked by fever. The initial convulsions may be general, but more frequently they are unilateral; when the left hemisphere is the seat of the lesion aphasia may be a symptom, and it is one of the slowest to disappear. The duration of the palsy is variable; sometimes recovery is very prompt, occurring in a few days; in other cases several months may elapse; and in still others the paralysis may be permanent. It is always most intense and widespread at first, and then slowly disappears, the leg usually showing its effect longest. The palsy, as indicated by the name of the disease, is a hemiplegia; in rare cases it is bilateral, due to a bilateral brain lesion. The affected limbs are at first limp and flaccid; as power returns contractures begin, and eventually there may be some spasm, with or without clonic movements. The portions permanently paralyzed become, as the opposite side grows, shortened and somewhat wasted. Sensation is unimpaired. The mind is usually defective when palsy is permanent,

and idiocy and epilepsy are not infrequent sequences. The prognosis is good as regards life, but guarded as to the degree and duration of subsequent paralysis. Repeated convulsions render the prognosis grave. The rapidity with which consciousness is regained and the palsy begins to disappear is an index of the rapidity and degree of final recovery.

Acute Softening.

Acute softening of the brain is the result of embolism or thrombosis. The most common cause of embolism is a recent endocarditis with vegetations upon the valves. Thrombosis occurs in atheroma and in syphilitic inflammation of the cerebral arteries. It may also occur in general diseases, acute or chronic, which produce systemic weakness or weakness of the heart.

The symptoms resemble more or less closely those of cerebral hemorrhage.

Embolism is to be distinguished by the age of the patient; it is most common from adolescence to middle life; whereas hemorrhage is more common after middle life. The onset is sudden and apoplectic in character. It is marked by coma and convulsions, but loss of consciousness is not usually so profound or of so long duration as in hemorrhage. This depends, however, somewhat upon the size of the vessel plugged.

Thrombosis differs from both hemorrhage and embolism in being more gradual in onset. Premonitory symptoms, consisting of headache, dizziness, and paræsthesia, are common. Consciousness may or may not be lost, depending upon the size of the occluded vessel and consequent area of softening. Delirium may follow the primary loss of consciousness, particularly in atheromatous softening. A secondary rise of temperature is more common in softening than in hemorrhage, and it may amount to hyperpyrexia.

Aphasia, monoplegia, and recurring convulsions are, according to Gowers, more common in softening than in hemorrhage; and in the subsequent chronic stage disorders of movement, mental failure, and emotional mobility are also somewhat more common in softening. An entire absence of focal symptoms is rather more common in softening.

Abscess of the Brain.

Abscess of the brain is most frequently the result of chronic suppurative otitis media; and in such cases the symptoms of mastoid disease usually precede it. It may be the result also of injury or disease of the cranial bones, or be part of a septic process. It is most common in male adults between the tenth and thirtieth years.

The most important consideration in diagnosis is the existence or antecedence of a cause of abscess in association with inflammatory cerebral symptoms.

The symptoms of brain abscess depend upon the character of the pus and its seat; in some cases, particularly when the cause is traumatic, the symptoms are inflammatory and the progress of the case is rapidly to a

fatal issue in a few weeks; in others, after a period of indefinite cerebral symptoms, the abscess becomes latent; and in still other cases, particularly when there is a general disease, the cerebral mischief may be obscured.

The symptoms in abscess which runs a rapid course are those of meningitis, rarely associated with focal symptoms. In the early stage delirium, convulsions, and coma are uncommon; but coma, at least, appears later, and may be preceded by rigors. In these cases, however, the cause is most frequently injury or septicæmia; whereas in the great majority of cases, which are the result of ear disease, the abscess during its formative stage gives rise to no symptoms except headache and disordered or weakened intellect, and may remain latent for months or even years. It may cease to be latent gradually, but more commonly latency ends abruptly, and symptoms of cerebritis and meningitis, occasionally with focal symptoms due to tumor, occur. The most important symptoms are fever, vomiting, headache, convulsions, paralysis, optic neuritis, and coma. The headache is persistent, and is often worse at the seat of disease. Vomiting is associated with constipation, fever, and sometimes with rigors and sweats. Convulsions are usually general, and are accompanied with paralysis, most frequently a hemiplegia. Rigidity of the neck and retraction of the head are not usually present except when there is an associated basilar meningitis. Optic neuritis, according to Gowers, is less common than in tumor, but more common than statistics would indicate. Delirium and coma usually close the scene.

When the abscess runs an acute course, and there has been injury or an existing otitis media, it is to be distinguished from *meningitis*. This cannot be done unless there are focal symptoms and optic neuritis; but abscess may be suspected if rigors are associated with the other symptoms.

If cerebral symptoms develop suddenly after a period of latency, either from rupture of the abscess or rapid extension of softening, the phenomena are those of apoplexy. Abscess then can only be suspected when the previous history indicates a cause.

When pressure symptoms exist, abscess is to be distinguished from *tumor* by the history; by its relatively rapid development; by the occurrence of rigors and fever. Pronounced localizing symptoms are in favor of tumor.

Tumors of the Brain and Its Membranes.

Tumors of the brain and its membranes are twice as common in males as in females. The tubercular and syphilitic are the most common, and next in frequency gliomata and sarcomata. Gowers states that the tubercular and sarcomatous tumors (including glioma and myxoma) constitute about four-fifths of non-syphilitic brain tumors. The same author says that three-fourths of the tubercular tumors occur during the first twenty years of life, and one-half the whole in persons under ten years of age. They occupy preferably the cerebellum and cerebrum.

Headache, optic neuritis, vomiting, mental changes, and giddiness are

the most constant symptoms. The headache is constant, but subject to paroxysmal exacerbations; occasionally the headache is unbearable; it unfits the patient for all mental work, prevents sleep, and may induce great despondency. Optic neuritis is nearly always present, regardless of the seat of the tumor. Vomiting is more common when the tumor is at the base of the cerebrum or in the cerebellum. The most common form of mental change is a gradual decadence of mental powers, but there may be more or less marked mental aberration, and disorders of speech, consisting of a slow syllabic utterance perhaps oftener than of difficult articulation. The paralysis which occurs is usually a hemiplegia or a monoplegia, which develops gradually and is associated with contracture. Occasionally the palsy is bilateral.

Convulsions are common, and may be general, or commence in such a way as to indicate the seat of irritation, as in the foot or hand.

The course of brain tumors is generally slowly progressive to a fatal issue in from six months to two years. Syphilitic tumors offer the best prognosis, and it is possible for tubercular tumors to become quiescent and encapsulated. (See Cerebral Localization.)

Multiple Sclerosis.

Multiple, disseminated, or insular sclerosis is a chronic degenerative affection of the brain and spinal cord which occurs preferably before middle age and in persons of nervous heredity. Its most constant symptoms are loss of muscular power in the limbs, a choreoid, jerky incoördination, especially marked in the arms; nystagmus, vertigo, and scanning articulation. Disturbance of sensation is not characteristic of the affection, but it may be met with as irregularly distributed anæsthesia or as paræsthesia.

There may be contraction of the field of vision before optic atrophy is discoverable; the latter is often developed in one eye before the other. Other symptoms occasionally present are vomiting, palpitation, and apoplectiform seizures. The general health of the patient remains good, and in spirits he shows surprising contentment.

Toward the close of the disease there are bulbar symptoms, such as interference with respiration and deglutition.

The duration of the disease is variable and its progress is not steadily retrograde; there are periods when the disease appears to be stationary. As a rule, it lasts from two to six years, but may continue twice as long. The prognosis is fatal; but the probability of length of life is to be judged from the rapidity with which the disease progresses and the presence or absence of bulbar symptoms and of complications—such as disease of the kidneys or bedsores.

It is distinguished from *locomotor ataxia* by the fact that the incoördination is most marked in the arms and that the reflexes are exaggerated, not diminished or absent.

From general paralysis of the insane it is distinguished by the absence of mental changes; by the articulation being slow, but accentuated and scanning, whereas that of paretic dementia is hesitating and indistinct, owing to difficulty in pronouncing certain consonants and to spasm of

the tongue and lips; by the absence of tremulousness about the mouth as seen in paretic dementia, and of the hallucinations and morbid impulses of the latter. Pupillary symptoms are less common in sclerosis than in general paralysis of the insane.

From *paralysis agitans* it is distinguished by the irregularity of the incoördinated movements and by the fact that they cease when the patient is at rest; whereas in paralysis agitans the movements are constant, rhythmic tremors. Moreover, in the latter the characteristic defects in articulation are wanting, and so are mental changes.

Glosso-labial-laryngeal Paralysis.

Glosso-labial-laryngeal, chronic bulbar, or progressive bulbar, paralysis is a chronic degeneration of nerve nuclei in the medulla, occurring most frequently after middle life, and characterized by slowly progressive loss of the power of articulation and of deglutition, with atrophy of the muscles concerned. The earliest symptoms manifest themselves in the tongue; there is difficulty in pronouncing words containing the lingual consonants, particularly *l* and *t*. At first the difficulty is noticed only when the patient is fatigued, and it can be overcome by effort; but eventually it is uncontrollable. The patient also loses gradually the power to protrude the tongue. In a short time the lips begin to lose muscular power; the patient can no longer pucker them, as in whistling, and has difficulty in pronouncing words containing the labial consonants, particularly *p* and *b*. Eventually he is unable to close the lips, and saliva constantly dribbles from them. Before the condition of the tongue and lips reaches its fullest development the soft palate becomes affected, and subsequently the pharyngeal muscles. Paralysis of the latter, with that of the tongue and soft palate, renders deglutition very difficult; fluids tend to regurgitate into the nose, and solid substances and fluids find their way into the larynx. The condition of the patient is pitiable; the intellect is undisturbed, so that he is fully conscious of his condition; in fully developed cases the only sound he can make is from the larynx. The meaning of the sounds has, therefore, to be guessed with the aid of his gestures. Sensation of the affected parts is not impaired, though reflex action is lost. The patient is sometimes easily moved to tears or to laughter, and during such emotions the paralysis of the lower part of the face becomes very conspicuous.

Progressive bulbar paralysis is often found in association with progressive muscular atrophy, with or without spastic paraplegia.

The course of the disease is progressive to a fatal issue in from one to five years. There may, however, be periods of temporary arrest of the disease. Death occurs from exhaustion depending upon insufficient nourishment, from bronchitis or pneumonia excited by particles of food being inspired, or from failure of respiration or heart.

Chronic Hydrocephalus.

Hydrocephalus implies an excess of fluids within the skull, either beneath the dura or within the ventricles. The former is called external and the latter internal hydrocephalus.

Internal hydrocephalus may be congenital; may occur after birth as the result of occlusion, usually from inflammation, of the openings into the fourth ventricle, or it may occur without ascertainable cause. It is characterized by a progressive enlargement of the skull; mental weakness frequently verging upon idiocy, and associated with physical weakness, occasional febrile attacks, convulsions, and vomiting. The eyeballs are prominent; there is nystagmus and optic atrophy.

In the congenital form the disease is present at birth and the enlarged head may form a serious impediment to labor. The head continues to grow in size, and may reach huge proportions. The fontanelles remain open, the skull is very thin, and the frontal portion projects over the face. The disease may progress rapidly and end in death from convulsions or wasting in a few months or a year, or at some stage it may be arrested and the patient live to an old age—with, however, feeble intellect and physique, and liability to epileptic seizures.

In the acquired form the disease may develop at any age. Enlargement of the head is less constant, but is not rare, after childhood; in its absence a positive diagnosis is usually impossible. The general symptoms are the same as in the congenital variety. Life is not usually prolonged beyond a few years, and death may occur in as many months.

Functional Nervous Affections. Chorea.

Chorea occurs almost exclusively between the fifth and twentieth years of life, and is especially apt to occur about the age of puberty. It is nearly three times as common in girls as in boys, and its causation is influenced by a nervous heredity, by rheumatism, by the season of the year (spring), and by pregnancy. The most common immediate cause is fright.

It is characterized by muscular twitching and jerky movements, irregular in time and rhythm, and occurring spontaneously. They tend to increase in frequency and range. They are at first controllable by a strong effort of the will, but only for a short time. Voluntary movements of the affected muscles become spasmodic, jerky, and incoördinate. Muscular power is generally impaired, but not often to a very marked degree, and is very rarely lost. Electrical excitability is often increased. Sensation is unimpaired. The most common mental change is apathy, which may be so profound as to border on dementia.

The disease begins gradually, the spontaneous jerky movements appearing first most frequently in the hands or face; in children regarded as emotional and excitable the movements are apt to be overlooked until they become more pronounced. The hands are moved involuntarily, or, when a voluntary movement is attempted, this is exaggerated in force or rapidity. If the patient attempt to pick up an object, he may succeed at the first attempt by a rapid jerky movement, or his hand may be carried beyond the object and several efforts be necessary before the object is seized. Sometimes also the patient is unable to relax his grasp quickly. The mouth is drawn to one side or the eyes closed by spasmodic winking. The head also may be jerked forward, but the body and legs are not affected so often or to the same degree. By

degrees the movements increase in frequency and range, and in severe cases become so nearly continuous that rest and sleep are obtained with difficulty, and may be so violent as to result in severe injury to the patient.

The disease may be limited to one side (hemichorea), but more frequently one side is more affected than the other, and it is most intense in the arms.

In some cases there is moderate pyrexia. Heart murmurs may be hæmic, from anæmia; valvular, from mitral disease, or, very rarely, from aortic disease. Endocarditis is very common as a complication. The respiration is often irregular and the pulse accelerated.

The duration of the disease is usually under six months, but relapses are common. Recovery is the rule; but it is a grave complication of pregnancy, about one-fourth of the cases proving fatal.

For the detection of the rare cases of paralytic chorea in which loss of power is more conspicuous than spontaneous spasmodic movements, Gowers suggests that the hand be held above the head, an action which brings choreic movements distinctly into play. The same author declares that "as a rule, when a child between seven and twelve years of age is said to have gradually lost the use of one arm, the disease is chorea."

Paralysis Agitans.

Paralysis agitans occurs most frequently between the fiftieth and sixtieth years. A nervous heredity has some determining influence. It is excited in some instances by shock, by fright, or by great mental anxiety; injury, and the exhaustion of an acute disease may also act as exciting causes. It is characterized in its fully developed form by general muscular tremors, which are spontaneous and rhythmical, and are associated with muscular weakness and rigidity. It begins most frequently by tremor of one hand, the tremor extending to the arm, thence to the leg of the same side, then to the opposite arm, and being followed by muscular weakness and rigidity. A leg, however, may be attacked first, and weakness may precede the appearance of tremor. The tremor itself is a to-and-fro movement produced by alternate contraction and relaxation of opposing muscles, and it continues during rest. The rigidity of the muscles causes flexion of the fingers and hands and, to a less extent, of the knees. The head falls forward, and the patient's gait is that known as "festinating," short quick steps being taken in rapid succession in order to preserve the equilibrium.

The muscles do not waste until late in the disease, and even then the atrophy is rarely marked. The reflexes are usually normal. Dull pains in the limbs are common early in the disease, and later the constant movements cause weariness. A subjective sensation of increased heat in the affected parts is very common; it may alternate with sensation of cold, or the latter may be the more constant. Pain is absent and the mind is unaffected, except that it shares in the general weakness.

The disease progresses very slowly and may last many years, death generally being the result of intercurrent affections.

Tetanus.

Tetanus is an acute disease of the nervous system, the essential characteristic of which is persistent tonic spasm of the muscles of the jaws (*lock-jaw*) and of the spinal and trunk muscles. The disease begins with stiffness of the jaw, which steadily increases until, within a few hours, there is complete tonic spasm of the jaw. The neck muscles, and then those of the spine and trunk, become rigid, so that the body is arched backward and may rest upon the heels and head (opisthotonos). The facial muscles share in the spasm, and by their contraction produce a horrid, grinning countenance (*risus sardonicus*). The contracted muscles become painful, and there is also epigastric pain. The rigidity is persistent, but is interrupted by exacerbations in which the phenomena already described are exaggerated, and in addition respiration is embarrassed, the face becomes livid, the skin bathed in sweat, and the patient is further distressed with increased pain in the affected muscles. The body may be bent forward (emprosthotonos) or laterally (pleurosthotonos). The temperature is not constant. It may remain normal, be moderately elevated, or hyperpyrexia may be present, especially toward and after the close in fatal cases. The spasm ceases during sleep, but subsequently returns.

The disease is most frequently traumatic in origin, but it may be idiopathic. Trismus neonatorum and puerperal tetanus are names given to special varieties which occur in newborn children and in puerperal women. Tetanus is much more common in men than in women, and Gowers states that three-fourths of the cases occur between the ages of ten and forty. It is much more common in hot than in cold countries, though cold is an exciting cause.

In traumatic and puerperal cases the disease usually develops in from a few days to two weeks from the time of injury or childbirth or abortion. In newborn children it occurs usually during the first week. It lasts from two to six weeks, but may be fatal much earlier, or in rare cases last longer. The mortality ranges from 50 to 90 per cent.; death is usually the result of heart failure or asphyxia, and occurs during an exacerbation of the tonic spasm.

Tetany.

Tetany is an acute affection of the nervous system characterized by spasmodic contractures, generally especially marked in the hands. These tonic spasms may be intermittent or continuous, and may be preceded or associated with paræsthesia. The disease is afebrile, may occur at any age, but is most common before the twenty-fifth year, and its occurrence is aided by diarrhœa, cold, lactation and pregnancy, the acute infectious fevers, and excision of the thyroid; in rare instances it occurs in epidemics. Tingling, burning, itching, or pain often precedes the appearance of the spasm, which generally seizes upon one or both hands first and then upon the feet. The fingers are flexed at the metacarpo-phalangeal joints and the thumbs adducted; the other joints are fixed in extension. The arms are flexed, but not strongly, at the elbow,

and may be adducted. The feet are extended at the ankle, and inverted, while the toes are flexed. In mild cases the spasms are not more extensive, but in severe cases the muscles of the trunk, thorax, head, and face may be involved, with characteristic interference with function and distortion of features. The spasms may be intermittent, remittent, or continuous, and in severe cases are attended by cramp-like pain. Usually the spasms are intermittent, recurring at intervals of a few minutes or hours. As in tetanus, when very severe and extensive, respiration and heart action may be embarrassed, the temperature rise, and profuse sweating occur; but the paroxysms become by degrees less severe; though, unlike tetanus, they may appear first, or may persist during sleep. The nerves and muscles in the intervals are abnormally excitable, so that percussion or compression of them or of the corresponding arteries, or the application of electricity, readily excites spasm. Gowers states that it is the only affection in which anodal-opening tetanus has been observed in man.

The duration of the disease is from a few days to a few weeks, depending upon its severity and upon whether the spasms are continuous or intermittent; it may be prolonged beyond this time, especially when the cause is excision of the thyroid. Patients are liable to recurring attacks upon exposure to the exciting cause. The prognosis is favorable to recovery in the large majority of cases; but death may result from the combined exhaustion of the spasms and the causal disease (diarrhœa), and is most unfavorable in the form following excision of the thyroid.

Writer's Cramp.

Writer's cramp is the most important of a series of neuroses occurring in persons whose occupation necessitates prolonged use of a special group of muscles. According to the occupation of the sufferer it is common to speak of "telegrapher's," "pianoforte-player's," or "stone-mason's cramp." The pathology in each case is probably the same, and as the diagnosis is based upon the evidences of disability associated with the occupation of the patient, it will be sufficient to describe the symptoms present in writer's cramp with the understanding that they cover essentially what exists in the other varieties of occupation neuroses.

Writer's cramp occurs most frequently in males from adolescence to middle life, and a nervous heredity has some causal influence. Injury to the hand or arm sometimes brings on an attack, but the most important causal factors are excessive writing performed in a constrained position—the hand being fixed and the fingers making most of the motions. The same or a greater amount of writing performed by a shoulder motion rarely induces the cramp. Moreover, depression of the general health, especially by worry and anxiety, is liable to precipitate an attack. The characteristic symptom is a tonic spasm of the thumb and forefinger of the writing hand, less frequently of the other fingers, but sometimes involving, in severe cases, the hand and forearm, the spasm being brought on sooner or later after each attempt at writing, but not at first by other movements. The affection almost always comes on gradually; the act of writing becomes slow and labored, the

fingers no longer contract and relax readily, but are stiff, and occasionally impart to the pen an unexpected motion, as the result of which the writing becomes angular, uneven, and too heavy. The fingers and hand also ache from weariness. The spasm tends to increase in intensity and range, and the writing becomes correspondingly irregular, difficult, and painful, until in severe cases, when various makeshifts have failed, all writing is found to be impossible. In such cases other movements of the same muscles are usually defective, display some incoördination, and may be followed by spasm. Tremor is rare. The muscular power is also apt to be impaired, but not to a great degree.

In other cases the patient can draw, sketch, or play the piano, but cannot write.

Pain is generally present, and sometimes is a prominent symptom; usually it is dull and aching in character, but it may be neuralgic and show a greater disposition to extend than the motor symptoms. The disease is curable if taken in time and if the patient can cease writing, but the duration will depend upon the severity of the affection and the general strength of the nervous system. The affection is liable to recur unless the bad habit of writing is overcome.

The diagnosis from degenerative affections of the brain or spinal cord, such as general paralysis of the insane and disseminated sclerosis, in which cerebral disease is sometimes first manifested by weakness and incoördination in the delicate movements of the hand, is to be made by noting the fact that, in the degenerations, other acts beside that of writing induce it from the very beginning, and that there are general as well as local symptoms.

From neuritis it is distinguished by the cause, the mode of onset, the presence of spasm, and the absence of the shooting pains and tenderness along nerve trunks characteristic of neuritis.

Epilepsy.

Epilepsy is a chronic disease of the brain characterized by sudden convulsive seizures which are first tonic, then clonic, are brief in duration, accompanied generally by complete loss of consciousness and often by cyanosis, and occur apart from organic brain disease, toxæmia, or other obvious cause. The attacks are not followed by any motor or sensory palsy, nor usually by any mental disturbance beyond drowsiness.

The disease may occur at any age, but the great majority of the cases occur before the twentieth year. Epilepsy or insanity in the parents predisposes to it. Exciting causes are convulsions during teething, especially in rickety children, worry, fright and anxiety, acute disease, particularly scarlet fever, and injuries to the head.

The attacks may be severe (*grand mal*) or mild (*petit mal*). The severe attacks are marked by loss of consciousness and falling to the ground, if the patient be standing. The muscles are first fixed in tonic rigidity, the eyes open, the face pale, and respiration embarrassed, with conjugate deviation of head and eyes. Soon the cyanosis lessens, the convulsive movements become clonic instead of tonic, respiration comes

in noisy puffs, and by degrees the patient falls into a heavy sleep or wakens to complete consciousness, but generally suffers with a headache for a while afterward. It is very common for patients to have warning (*aura*) of the approach of an attack. This may consist of flashes of light before the eyes, or of a sensation or motion in the arm, face, or leg. Sometimes also the convulsion begins in one arm or in the face. During the convulsion, biting of the tongue and involuntary discharge of urine are common and very characteristic symptoms. The convulsions recur at very irregular intervals; and many occur in one day, or they may be delayed for weeks and months. Moreover, they may be diurnal or nocturnal, and when exclusively nocturnal they may escape the detection of the patient and his friends for years.

When they are suspected, inquiry should be made as to the existence of a bitten tongue, of subconjunctival hemorrhage, or of nocturnal enuresis.

Rarely there is a series of convulsions uninterrupted by intervals of consciousness (*status epilepticus*).

After attacks patients are conscious, but usually dull and drowsy; sometimes they are very quarrelsome, and may commit acts of violence.

In the milder cases, classed as *petit mal*, the seizures vary widely in symptoms, but they consist in general of a disturbance of sensation, less frequently of motion, associated with a partial loss of consciousness. The patient becomes momentarily giddy and faint, sits down or grasps an object for support, a mist comes before his eyes, and he loses consciousness partly or completely, but only transiently. Slight convulsive movements may occur, but are not usual.

Urine may be voided unconsciously. The patient remains dazed for some moments after the attack and may commit strange actions; that of undressing is said to be one of the most frequent.

The duration of the disease is very uncertain. When it has existed for several years it is rarely cured. The best prospects of cure are in those over twenty years of age, when the fits occur at long intervals and when treatment can be kept up continuously. Death does not often occur during a convulsion, but fits occurring in dangerous places not uncommonly lead to accidental death. Life is shortened by it, but it is difficult to say to what extent.

Hysteria.

The manifestations of hysteria are seen in permanent symptoms, known as the stigmata, and in the hysterical attacks.

The *stigmata* are often detected only by special examination. The patient is not usually cognizant of their presence.

1. Sensory Anæsthesia. The sensibility of the surface and the special senses are affected. *Analgesia* is common. It may be general or limited to an arm or a leg, or to areas on the limbs or trunk. Other forms of cutaneous sensibility may not be affected at the same time, although any variety of anæsthesia may occur. The muscular sense may be lost. Eye symptoms, due to disturbance of the sensibility of that organ, are common. Vision may be distinct or dim. Limitation of the

visual field is common, and most characteristic. Achromatopsia, alteration of color sense, is common in hysteria. Diminution of hearing and loss of smell and taste are common sensory symptoms in this affection.

Hemianæsthesia is a common symptom of hysteria. One-half of the body seems to have lost consciousness. The skin bleeds in small amounts when wounded. The mucous membranes are affected, as the conjunctiva, half of the buccal cavity, and the tongue. The muscular sense is lost. There is diminution of the sense of hearing on the affected side, and loss of the sense of taste and smell in the corresponding positions. Amblyopia, or amaurosis, occurs in the eye of the corresponding side.

In hysteria, hyperæsthetic regions are of common occurrence. These areas or "hysterogenous zones" are important manifestations. The sensitive points are tender on pressure, although when the patient's thoughts are diverted firm pressure is not observed. The hyperæsthetic areas are often the seat of pain.

These areas may be extensive or quite circumscribed. They are most common in the head and trunk, on the sides of the chest, under the breast, and on the sternum. Hyperæsthesia of the spinal column and of the lower abdominal region is of very common occurrence. The whole spine or small portions only of it are affected. A slight pressure may cause severe pain. Hyperæsthesia of the eye, ear, and other senses occur.

2. *Hysterical Paralysis.* A frequent manifestation of hysteria is paralysis of one or more groups of muscles. It may occur suddenly or come on gradually. The paralysis is of central origin, due to loss of the power of will to effect contraction of the muscles. The following muscles in order of frequency are affected: 1. The muscles of the lower limbs. 2. The vocal cords. 3. The pharynx and œsophagus. 4. The muscles of the arms. Hysterical paralysis of the facial muscles does not occur. In paralysis of the lower linbs the patient may be able to move the legs in bed, but cannot walk. Both flaccid and spastic paralyses are seen in hysteria. The tendon reflexes may be exaggerated.

3. *Hysterical Contractures.* Contractures occur alone or with anæsthesia or paralysis. They may be temporary, but often become permanent. In the hands and feet there are flexor contractures. Extensor contractures are more common when the muscles of the large joints are affected. They often follow a convulsion and may be limited to one entremity, to the extremities of one side of the body, or the lower extremities alone.

4. *Vasomotor Disturbances.* The surface of the skin may be cool and pale, or hot and red. The two conditions may alternate. The affected portion is limited to an extremity, or to the skin about a joint. Among other vasomotor disturbances frequently seen, hemorrhages from internal organs take place. Hæmatemesis, hæmoptysis, and other bleedings may be found.

Hysterical *fever* has been observed. The increased temperature occurs at the time of an attack of hysteria. Care must be taken that the rise of the mercury is not produced by rubbing and pressing the ther-

mometer by the patient. The temperature should be taken in the rectum. Modifications in the secretory organs are common. The perspiration may be increased or absent. The flow of saliva is similarly modified. Ischuria or diminished secretion of urine is often seen. Polyuria of hysterical origin is more common. The urine is light in color and of low specific gravity.

5. *Visceral Symptoms.* The most common perversions of the functions of internal organs are seen in those belonging to the gastro-intestinal tract. They have been fully dealt with in the section on diseases of these organs. In addition to the manifestations mentioned, hysterical tympanites is of common occurrence. The accumulation of gas simulates tumor, pregnancy, or peritonitis. Under anæsthesia the hysterical tumor is dissipated. The gas may be removed by a rectal tube.

In other portions of this work reference has been made to the cough of hysteria, and to a peculiar form of pulmonary hemorrhage seen in hysteria. Increased frequency of respiration, modification of the normal rhythm, and dyspnœa, usually unattended by distress and with normal pulse, are frequent phenomena of pulmonary hysteria.

In cardiac hysteria increased frequency of the heart's action on the slightest emotion, with or without precordial distress, is common.

Hysterical or pseudo-angina often occurs. Flushes, both general and local, are common symptoms. The joints are frequently affected in hysteria (see p. 143).

The *Mental Constitution.* The characteristic of the patient most paramount is selfishness. The various forms of expression of feeling are almost always excited by the desire of the patient to attain some object. The patients are irritable and emotional. They are easily depressed, extremely sensitive, and subject to violent emotional expressions. They exaggerate their sufferings, do everything to command attention, and attempt to excite sympathy. Any desire that is to be accomplished is secured by sly means, to say the least, or actual deception. The will-power is lost entirely or enfeebled. The patients are usually bright and vivacious, or emotional in turn. Mental characteristics may be absent entirely.

The general nutrition may not be affected, although the ill nourished and weakly are more often hysterical.

Hysterical Attacks. A so-called attack of hysteria may be the first manifestation of the disease, or the patient may not become subject to such attacks until some time after the permanent stigmata have developed. The attacks may be made up of subjective symptoms only, the patient complaining of vertigo, anxiety, precordial or respiratory distress, a sense of fulness in the throat, or a lump in the œsophagus (globus hystericus). The objective symptoms of hysteria are seen in aberrant displays of emotion or convulsive movements, with or without a loss of consciousness. *Convulsions* may be preceded by emotional disturbance, or by painful sensations in the chest or abdomen. In the minor convulsions the movements are clonic and irregular. Each series of convulsive movements lasting a few minutes, is followed by an emotional attack, when consciousness, which was lost, is restored.

Instead of convulsive movements the patient may fall into a relaxed state with unconsciousness. At the time of the attack the abdomen may be distended with flatus. Urine light in color is passed in large amounts afterward.

Hystero-epilepsy is the most exaggerated convulsive form. The attack may come on suddenly, or be preceded by milder hysterical symptoms. Areas of hyperæsthesia are often detected at this time. They are more marked over the ovaries and the upper dorsal vertebra. The attack is preceded by the globus or by a feeling of extreme oppression. The attack is divided into four stages. In the first stage an epileptic paroxsym is simulated. The convulsions, which are at first tonic, are followed by gradual relaxation and coma. The duration of the attack is longer than epilepsy. Following the convulsions there is a violent display of emotion with contortions; and cataleptic positions are assumed. In the third stage peculiar attitudes are assumed which express the various passions. This period is followed by a return of consciousness, with delirium and hallucinations. This, the fourth stage, may continue for several days. The attacks may recur for days, followed by a trance-like state which in turn may continue for a long period.

The diagnosis of hysteria is based upon the presence of the stigmata, the peculiar character of the pain, the occurrence of emotional attacks, and the globus hystericus. The pain and other subjective symptoms are influenced by suggestion. The paralyses are usually associated with anæsthesia. They are always variable. All forms of organic paralyses may be simulated by hysteria.

Neurasthenia.

The symptoms may be general or local or may be combined. The patient is usually under weight, and more or less anæmic. Debility may be so marked as to compel the patient to remain in bed. Local neurasthenia is expressed in cerebral, spinal, cardio-vascular, gastric, and sexual forms. In cerebral neurasthenia there is sensation of weight and fulness in the head, with flushes. The patient may be drowsy, and is usually irritable and depressed. Headache may be complained of. The pain is most common in the back of the head or the neck. Neck weariness is a common symptom. Any mental effort, even of the smallest degree, is accomplished with difficulty. The smallest amount of mental work may require a painful effort. The patient is likely to complain of aching and weariness of the eyeballs after reading a few minutes. Flashes of light are often present.

The symptoms of spinal neurasthenia are those of what was formerly termed spinal irritation. Local tenderness is found all along the spine in small areas. In the cervical spine aching is common. The patients weary on the slightest exertion, and are subject to backache and aching pains in the legs.

Cardio-vascular symptoms, as palpitation of the heart, irregularity, increased frequency, and precordial pain are common. Vasomotor symptoms are most pronounced. Flushes of heat and transient hyperæmias are frequently seen. Sweatings may occur. Arterial

throbbing is very common. The capillary pulse can often be seen. Throbbing of the aorta and of the carotids are most common in neurasthenia.

The gastro-intestinal symptoms have been discussed in Chapter V.

Neurasthenia is frequently associated with lithæmia.

Pain in the Head.

Pains in the head may be classified according to location into those due to affections of the *scalp*, those due to affections of the *cranium*, and those due to *intra-cranial* conditions.

I. Affections of the *scalp* are to be further classified as those of the skin, those of the occipito-frontalis muscle, and those of the nerves. The occurrence of itching and burning commonly indicates some local condition of the skin; if the itching is slight seborrhœa should be looked for; if more severe, eczema; and burning and itching of a severe type commonly indicates dermatitis venenata; the pediculus capitis should not be forgotten. A feeling of tension with soreness accompanies the eruption of erysipelas. Intense local irritations are caused by burns and scalds, the latter, however, because the hair is not immediately destroyed, is alone likely to give rise to error. A sore feeling with local tenderness, limited to a swelling sharply delimited, with a sensation of less resistance in the centre and some darkening of the skin is diagnostic of a bruise. Hyperæsthesias of the scalp frequently accompany meningeal and cranial affections, and there are even local changes, such as the so-called puffy tumor of necrosis of the inner table of the skull.

Sharp pains in the occipital or frontal region, increased by wrinkling the scalp, or brief pressure, but generally relieved by firm and constant pressure, occurring with irregular periodicity, and associated with meteorological changes, are suggestive of occipital *myalgia*. The diagnosis is confirmed by the presence of other symptoms of lithæmia.

The sensory *nerves* of the scalp and face are the trigeminus and the branches of the cervical plexus. The distribution is as follows: the ophthalmic division of the trigeminus is distributed to the eyeball, lachrymal gland, the mucous membrane of the nose and eyelids, the integument of the nose and upper eyelid, the forehead and the anterior half of the hairy scalp. The superior maxillary division supplies the skin over the malar bone, and that of the lower eyelid, side of the nose, and upper lip; the upper teeth, the upper part of the pharynx, the antrum of Highmore, and the posterior ethmoidal cells; the soft palate, tonsil, and uvula, and the glandular structures of the roof of the mouth. The inferior maxillary division is distributed to the side of the head, the upper anterior portion of the external ear, the external auditory canal, the lower lip, and lower part of the face; the tongue, the mouth, the lower teeth and gums, the salivary glands, and the articulation of the jaw. The great occipital is distributed to the back of the head, the small occipital to a narrow region just in front of it, and the

great auricular to the skin of the posterior portion of the pinna and the skin over the mastoid and parotid gland.

Neuralgia occurs in the form of paroxysms of pain accurately located in the course of one or more of the nerve trunks, and presenting points of special sensitiveness where the nerve emerges from the skull, and where it divides for its cutaneous distribution. The pain is usually relieved by firm pressure, but it is to be remembered that sharply localized pressure on the nerve trunks against the hard skull will cause a traumatic tenderness. The character of the pain is variable: it may be of the most acute or rending form, or, but more rarely, a persistent dull ache; it may be throbbing, or in successive paroxysms at brief intervals or regularly periodic. There are often associated vasomotor, secretory, and motor disturbances; and local blushing or sweating may be observed along the course of the nerve; and spasms may occur in the muscles, as of the eyelid, or more general as in the terrible tic douloureux, distinguished by the pain from tic convulsif. The commonest seats are the supra-orbitals, the dentals, the auricular branches, and the occipitals; in the great majority of cases it is unilateral.

Pain, undistinguishable from neuralgia, is frequently due to some *local irritation;* foreign bodies have been known to cause paroxysmal attacks for a number of years, until removed; diseases of the bones are a prolific source, especially in the case of the jaws and the cervical vertebræ. Enlarged cervical glands occasionally irritate the great auricular or small occipital. Bilateral occipital pain is very characteristic of cancer of the cervical vertebræ. In these cases there is usually pain upon movements of the head or pressure upon it, and some stiffness of the neck. Intra-cranial growths occasionally cause pains, usually paroxysmal, limited to one of the branches of the trigeminus.

Certain of the cephalic nerve pains are symptomatic of disturbance of the associated but distant nervous distribution. Pain in the region supplied by the ophthalmic division is very common in influenza. It is usually dull, aching, and continuous, increased by pressure and anything tending to increase congestion. A severe acute attack of indigestion will produce ocular and supra-orbital pain. Refractive lesions of the eye cause the same character of pains, but increased by using the eye and relieved by rest and atropine. The use of the latter is an important diagnostic procedure. Pain in the temporal region and the external auditory meatus is often due to intense irritation of some of the branches of the inferior dental; the usual cause is cancer of the tongue, but irritable lingual ulcer may also produce it, and even severe inflammatory conditions of the lower jaw. The pain is described as sharp and lancinating and paroxysmal, liable to exacerbations, especially when the primary lesion is excited, and relieved when it is alleviated. Pain may be caused in the ear alone, when there is irritation of the teeth.

Perhaps in the majority of cases of cephalic neuralgias the cause is to be found in some systemic disturbance. If the attack is preceded by a desire to sleep, occurs when the dew-point is high, and is associated with increase of urates in the urine, it is probably *lithæmic;* the pure gouty

forms are more apt to succeed indulgence in rich food or heavy meats and there is ordinarily irritability of temper. *Diabetic* neuralgias invariably are worse as the amount of sugar excreted is increased, and there are usually similar affections of the nerves in other parts of the body. Regularly periodic pains, worse in the spring and fall, occasionally preceded by a slight chill or malaise, suggest chronic *malaria*. The diagnosis can readily be confirmed by examination of the blood, and detection of enlargement of the spleen. *Syphilitic* neuralgias are usually worse at night; the pain is described as boring, and may indeed simulate periodicity. There is apt to be some thickening of the bones, and perhaps a diminution of elasticity of the tissues, and almost always local tenderness. The pain is almost immediately improved by potassium iodide. In *anæmic* neuralgias the pain is not characteristic, but they are temporarily improved by the recumbent posture and stimulants, and is worse during menstruation. The general appearance of the patient and an examination of the blood readily suggest the cause. In *locomotor ataxia* there are occasional cephalic crises of neuralgic nature; these come on suddenly and are exceedingly severe, but usually occur only at long intervals; the pain is shooting or stabbing and does not remain located to one nerve trunk. Chronic *lead* and *alcohol* poisoning also cause neuralgias, but they are not of themselves characteristic and never occur as isolated symptoms, being frequently associated with peripheral neuritis of other parts.

Dull burning pains commencing perhaps with a chill, and accompanied by febrile symptoms, indicate *inflammations* of the mucous membranes of the head. A dull persistent headache located just beneath the eyebrows often accompanies coryza and indicates extension to the frontal sinuses; if the nose alone is involved there is a feeling of fulness and occasional sharp pains or tickling sensations. A feeling of dryness and some discomfort on swallowing accompanies the various forms of stomatitis and pharyngitis; in the latter there is also a sensation of tickling and fulness in the ear, due to extension along the Eustachian tube. Pain at the angle of the jaw, with tenderness and increased on swallowing, almost invariably unilateral and associated with swelling of the parotid, is unmistakably parotitis. The neuralgias and inflammations of the middle ear are exceedingly painful; they may consist of a sharp continuous pain, or a series of regular exacerbations and remissions, or a throbbing sensation; often pain radiates to the jaws and side of the face. As suppuration occurs the feeling becomes one of extreme tension until the membrane is perforated, when there is immediate relief. Tinnitus throughout the whole course of the case is very common. The inflammations of the eye produce local pain, usually causing the sensation of a rough foreign body. Usually there is slight supra-orbital tenderness, and in iritis sharp pains radiate over the whole area of distribution of the two upper branches of the fifth. Certain ulcers of the mouth are comparatively painless; noma often developing insidiously; while syphilitic ulcers are to be distinguished by their painlessness from simple and tubercular ulcers, which are very irritable, and carcinomata, which are liable to paroxysms of pain even when not irritated.

It may not be out of place to mention the value of certain anæsthesias as diagnostic signs; thus in neuritis of branches of the fifth, there may be cutaneous anæsthesia while there is tenderness over the nerve trunks.

II. *A dull, constant headache,* limited to a small area, later increasing in severity, and the pains assuming, perhaps, a boring character; tenderness, often very severe, over the affected area, and probably slight œdema of the scalp, with some rigidity of the muscles of the neck, and the ordinary signs of the inflammatory process, indicate inflammation of the cranial *bones.* In the simple cases there will usually be some history of injury, the pains will not be especially periodic, and the fever irregular. In the syphilitic cases there will be the history and symptoms of infection, the pain will become especially worse at night, and usually there will be concomitant rise of temperature. The pains will also be controlled by potassium iodide, but as it often requires enormous doses to accomplish this result, the failure of a moderate dose should not be considered as exclusive.

III. Intra-cranial Headaches. Intra-cranial headaches are functional or organic. Both forms may be acute or chronic. The typical acute functional headache is seen in the more or less common type known as *migraine* or *hemicrania.*

Migraine is a periodical neurosis characterized by pain affecting the trigeminus and other cranial nerves. The headache is usually unilateral, and while probably due to vasomotor disturbances is always associated with vasomotor symptoms. It occurs more particularly in women, frequently begins in early childhood and continues throughout adolescence. It is often hereditary. It occurs most frequently in women who suffer from anæmia or from menstrual difficulties. The habit which predisposes to the headache may develop after long physical or mental over-exertion. The attacks, however, are excited by over-exertion, mental excitement, or disturbances of digestion. Pain of migraine is possibly situated in the pia and dura mater.

Symptoms. The attack develops with or without premonitions. In each individual different prodromal symptoms are recognized as indicating the approach of an attack. Undue nervousness, a general sense of discomfort, pressure or heat in the head, vertigo, tinnitus, spots before the eyes, excessive yawning, or repeated chilliness are the most common.

Premonitory Symptoms. The pain is most frequently felt on the left side of the head first. It is seated in the anterior frontal, the temporal, or parietal regions. The pain is continuous and increases in intensity to the height of a paroxysm. Painful points are not usually detected, although the whole skin may be hyperæsthetic. The patient is sensitive to light and sound, intolerable nausea intervenes, and vomiting may occur at the height of the attack. The eye symptoms are very pronounced. Flashes before the eyes, scintillating scotoma, or hemianopia may occur.

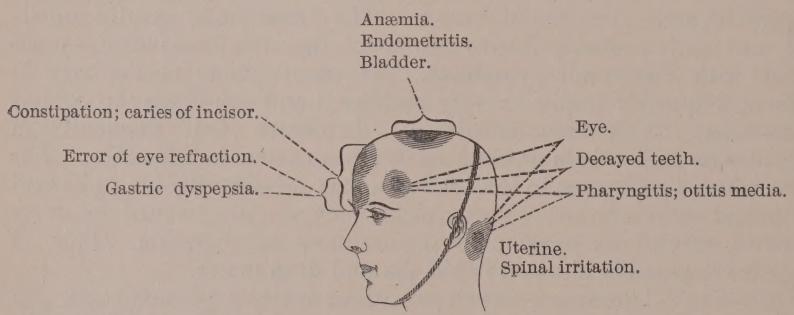
The vasomotor symptoms that attend the attack are of two varieties, causing the disease to be divided into the spastic and angio-paralytic forms. In spastic migraine the skin on the affected side is cool, the forehead and ear pale, the temporal artery is contracted, the pupil

is dilated, and the flow of saliva increased. In the paralytic form there is redness of the face on the affected side. The temporal arteries are dilated and pulsate strongly. The face is hot, the pupils contracted, and there is often unilateral sweating.

CHRONIC HEADACHES. Chronic headaches of functional origin are usually habitual as to the constancy of attacks, although the period between the attacks may vary. The nerves affected are the trigeminus, the four upper cervical and sensory branches of the vagus to the posterior fossa of the skull. Three types of such head pains are seen: ordinary headache, migraine, and neuralgia. Headaches are caused as a rule by diffuse irritations located in or referred to the peripheral ends of the nerve tracts above referred to. Neuralgias, on the other hand, are caused by irritations of the trunks of these nerves.

Causes. 1. Hæmic. *a.* Anæmia. *b.* Diathetic states (gout, rheumatism, diabetes). *c.* Infections (malaria, syphilis, specific fevers). 2. Toxic (lead, and other mineral poisons, alcohol, uræmia, tobacco). 3. Neuropathic states (epilepsy, neurasthenia, chorea, hysteria, neuritis). 4. Reflex causes (ocular, naso-pharyngeal, auditory, gastric, sexual, uterine). 5. Organic disease.

FIG. 162.



Showing the location of pain in various headaches. (After DANA.)

Headaches are divided according to their situation into frontal, occipital, parietal, vertical, diffuse, and combinations of both. The most common forms are the frontal, the frontal-occipital, and the diffused. Ocular headaches are usually frontal, when due to errors in refraction. When due to muscular insufficiencies they are occipital and cervical. Naso-pharyngeal headaches are dull frontal or diffused. When the pharyngeal tonsil is enlarged the headache may be dull, frequently recurring and seated in the occipital region. In follicular tensillitis the headaches are diffused. In obstruction of the Eustachian tubes they are diffused. In disease of the middle ear they are temporal and occipital. Gastric or dyspeptic headaches without constipation are often occipital, sometimes frontal. With constipation and intestinal irritation they are diffused and frontal. Uterine and ovarian headaches are occipital and vertical. *Neuropathic* headaches are seated on the top of the head, as in clavus, or are associated with spinal irritation. Neuras-

thenic headaches are usually associated with a sense of pressure or weight, and are seated in the frontal and vertical regions. In spinal irritation the pain is of a boring character in the occipital region. The earliest symptom of the neurasthenic headache is neck weariness and pain in the neck. The neurasthenic headaches occur in brain-workers, when the brain and eyes are overtaxed. Headaches in epilepsy are severe, and are confined to the vertical or occipital region. *Organic* headaches are usually violent, associated with fulness and throbbing. They may be remittent, becoming more intense with each exacerbation. The organic headaches may be due to inflammation, to abscess and softening, to tumor, to congestion of the brain, and to inflammations in the meninges. Anything which increases the blood will increase the pain in organic headaches. In acute inflammation of the brain the pain is agonizing, continuous, associated with vomiting and fever, and sometimes delirium. In abscess of the brain the pain is less violent. It is occasionally paroxysmal and attended by paralyses and disturbed intellection. In tumor of the brain the headache is severe and paroxysmal. In congestion the pain is dull, increased by stooping, by sleep, and by bodily or mental fatigue. Some congestive headaches are due to violent exercise and relieved by bleeding at the nose. In all congestive headaches the face is flushed, the bloodvessels are turgid, and the vessels in the eye-ground will be found to be overloaded. In meningitis the pain is constant, is more or less fixed, and sometimes very sharp. Syphilitic headaches are frontal or temporal, worse at night, and often periodic.

Headaches are divided according to the *character* of the pain: 1. Pulsating and throbbing. 2. Dull and heavy. 3. With constriction, squeezing, or pressing. 4. Hot and burning. 5. Sharp and boring. The headaches of the first class are usually associated with vasomotor disturbances, as in migraine; to the second class belong the toxic and dyspeptic headaches; to the third the neurotic and neurasthenic; to the fourth rheumatic and anæmic; to the fifth hysterical, neurotic and epileptic. *Vertigo* is a common accompaniment of the dyspeptic type of headache situated in the frontal regions. *Somnolence* is more marked in the syphilitic, anæmic, and malarial headaches. *Nausea* is more common in occipital forms of headache.

Duration. Eye-strain causes occipital pain, which is rarely persistent, but comes on after prolonged use of the eyes. It may be associated with headaches in other parts, due to other causes. In chronic meningitis the headache is persistent and located in the vertex or the parietal regions. When thickening of the meninges with adhesions take place from trauma there is constant pain, sensitiveness of the head, incapacity for study, and frequent exacerbations of the pain. Uræmic headache is inconstant. Persistent headache may in the latter stages of Bright's disease be present. In diabetes persistent headache occurs. In atheroma pain in a part or the whole of the head is common. It may be persistent though subject to exacerbations in case of excitement or violent exercise. Headache following study in children is due to brain strain, to the eyes, or to indigestion. Persistent headache is sometimes due to asthma. In rare instances headache is said to be idiopathic.

Neuralgic headaches are usually periodic and may be associated with throbbings or pulsations. They are associated with vasomotor signs. Hysterical headaches are irregular and shifting; they persist after all causes are removed; they are replaced by pain in other parts of the body. They are usually associated with other manifestations of hysteria.

INDEX.

ABDOMEN, aspiration of, contra-indications to, 160
color of, 473
diminution in size of, 473
general enlargement of, causes of, 470
palpation and percussion of, 474
local enlargement or tumors of, 472
markings on, 473
method of marking surface of, 468
movements of, 473
palpation and percussion of lower quadrants of, 475
peristaltic movement seen through walls of, 474
point for puncture of, 160
quadrants of, contents of, 468
shape of, 473
superficial veins of, 474
tumors in, 478, 479, 480
tumors of, epigastric pulsation in, 368
walls of, in peritonitis, 475
pain due to disease of structures of, 469
Abscess, fæcal, 477
gouty, 714
pelvic, 476
pericæcal, causes of, 572
distinguished from appendicitis, 571
perigastric or subdiaphragmatic, 479
perinephritic, 680
distinguished from appendicitis, 571
præcordial, 386
retro-pharyngeal, 460
stitch, cause of, 163
subphrenic, 337
Acid, hydrochloric, in gastric contents, tests for, 490
uric, 662
Acetonæmia, 668, 718
Acetonuria, 648
Acromegalia, 65
Actinomyces, 164
in sputum, 281
Actinomycosis, 772
Acute peritonitis, facies in, 118
Addison's disease, 699
bronzed skin in, 74
pigmentation of buccal mucous membrane in, 431
tongue in, 439
Adenitis, diagnosis of, 134
Ægophony, 263, 330
Age, in ætiology of disease, 24
influence on temperature, 102
Albuminometer, 641
Albuminuria, causes of, 642
functional, 642
in tonsillitis, 455
Alcoholism distinguished from apoplexy 839
Amaurosis, uræmic, 667
Amblyopia, tobacco, 807
Amœba dysenteriæ, 553
in fæces, 539
in pus, 162, 166
in sputum, 275
Anæmia, 688
cardiac murmurs in, 366
classification of, 689
clinical divisions of, 172
from hemorrhage, 690
hemorrhage in, 78
idiopathic or pernicious, 691
in constitutional and local diseases, 690
in interstitial nephritis, 675
in nephritis, 670
local, 173
Mackenzie's rule for detecting, 132
murmurs in, 381
neuralgia in, 856
œdema in, 93
parasitic, 689
splenic, 697
toxic, 689
venous hum in, 384
Anæsthesia, cerebral, 789
in hysteria, 850
of skin, 788
spinal, 789
Anasarca, 92
cardiac, 95
of Bright's disease, 95
Aneurism, aortic, 421
bronchorrhœa in, 423
conditions with which confounded, 428
confounded with phthisis, 423, 428
diagnosis of, 427
general symptoms of, 422
physical signs of, 425
præcordial pain in, 386
symptoms of, 422, 423
tracheal tugging in, 427

Angina Ludovici, 449, 460
pectoris, arterial tension in, 373
associated with arterial sclerosis, 421
cardiac lesions with which associated, 387
diagnosis of, 387
false, 388
in aortic incompetency, 405
pain due to, 387
Angle of Ludwig, 228
Anidrosis, 77
Anilines, basic, 153
Ankle clonus, 793
Anorexia in gastric neuroses, 515
Anthrax, 774
bacillus of, 165, 775
Antrum, abscess of, 199
lacrymal duct in, 200
obstruction of duct of, 200
Aorta, aneurism of (*see* Aneurism).
epigastric pulsation in, 369
atheroma of, cardiac murmurs in, 367
diseases of, pain in, 386
pulsation of, 368
confounded with aneurism, 428
Aortic incompetency, 404
arteries in, 406
general symptoms of, 405
pharyngeal hemorrhage in, 451
physical signs of, 405
pulse in, 406
thrill in, 406, 348
valvular murmurs in, 406
obstruction, 406
thrill in, 348
Apoplexy, 837
conditions with which confounded, 839
distinguished from embolic softening, 839
in interstitial nephritis, 676
ingravescent, 838
pain in extremities, forerunner of, 45
seats of hemorrhage in, 838
vomiting as symptom of, 500
Appendicitis, conditions with which confounded, 571
confounded with intestinal colic, 527
with typhoid fever, 733
distinguished from intestinal obstruction, 566
recurrent, 569
with perforation, 570
without perforation, 567
Appendix, diseases of, palpation and percussion in, 475
Apraxia, 798
Aprosexia, 458
Arcus senilis, 124
Arteries, auscultation of, 380
disease of, subjective symptoms in, 391
of the neck, pulsation of, 368
pulsation of 389
Arteries, sclerosis of, causes of, 419
symptoms and signs of, 420
Arterio-capillary fibrosis (*see* Arteries, sclerosis of).
Artery, tension of, causes of, 185
recognition of, 186
Arthritis, rheumatoid, acute, 710
chronic, 711
diagnosis of, 712
hand in, 127, 712
pulse in, 375, 711
senile form of, 712
skin in, 711
Articulation, occipito-atlantal, tuberculosis in, 121
Ascites, 578
in general enlargement of abdomen, 471
Aspiration, 159
of body cavities, 160
Asthma, 302
dyspnœa in, causes of, 285
Atavism, 28
Ataxia, 792
febrile, 107
hereditary, 830
Ataxic state, 107
in acute diseases, 108
Atelectasis, 326
Athetosis, 131, 791
Atrophy, optic, 807
progressive muscular, 831
hand in, 127
simple idiopathic muscular, 832
Auscultation (*see* Chest).
general section on, 53

BACILLI, 149
comma (*see* Spirilla).
of Booker, 547
Bacillus, anthrax, 775, 165
in blood, 703
coli communis, 162, 166, 544
comma, 545
Eberth's, 731
Klebs-Löffler, 757
lepræ, 778
Löffler's, 453
of glanders, 165
in blood, 703
of influenza, 166
in sputum, 281
of syphilis, 164
of tetanus, 166
tubercle (*also see* Sputum).
in fæces, 547
in blood, 701
typhoid fever, 547
Backache, 48
in fevers, 108
Bacteria, 147
cultivation of, 154
determination of specific nature of, 157
in pus, 161

Bacteria in the saliva, 432
pyogenic, 162
Bacteriology, apparatus necessary in, 151
preparation of, 151
collection of material in, 152
cover-glass preparations in, 153
culture media in, 154
"hanging-drop" in, 154
inoculation of animals in, 157
Koch's laws in, 146
methods of research in, 146, 150
of staining in, 153
preparation of cultures in, 156
special diagnosis in, 157
sterilization in, 151
Bacteriuria, 660
Bell-tympany, 262
Bile-ducts, cancer of, 604
Biliousness, 585
Blindness, functional, 807
Blood, alterations in, 172
counting corpuscles of, 685
cover-slip preparations of, 152
estimation of hæmoglobin of, 683
in gastric contents, tests for, 489
in leucocythæmia, 694
in stools, in diarrhœa, 530
naked-eye appearances of, 683
parasites in, 701
pressure, 185
proportion of red to white corpuscles of, 683
Body, general form and nutrition of, 61
Bones in general diagnosis, 139
nodes on, 139
Boulimia, 503, 515
Bradycardia, 375
Brain, abscess of, 841
acute softening of, 841
anæmia of, 836
hyperæmia of, 837
lobes of, 799
occipital, 799
parietal, 799
prefrontal, 797
localization of areas, 795
softening of, 839
tumors of, 842
distinguished from meningitis, 836
Brawny induration, 98
Breakfast, test, 490, 491
Breath, fœtor of, in pharyngeal affections, 454
Breathing, alteration of the rhythm in, 255
bronchial, 253, 256
in pleurisy, 330
varieties of, 257, 258
broncho-vesicular, 254, 258
feeble, 254
jerking inspiration in 256
prolonged expiration in, 256
puerile, 254
vesicular, 253
diminished or absent, 255
Breathing, vesicular, exaggerated, 254
Bright's disease, acute rhinitis in, 188
anidrosis and boils in, 77
erythema in, 88
facies in, 118
Bronchi, dilatation of, 300
diseases of, recognition of, 294
obstruction of, 302
Bronchiectasis, 300
Bronchitis, acute, 295
capillary, 297
chronic, diseases with which associated, 298
collapse of lung in, 326
fibrinous, casts in, 272
fœtid or putrid, 299
plastic, 299
specific, 300
Brownian movement, 154

CACHEXIA in gastric cancer, 508
malarial, 770
of carcinoma, 183
Cachexiæ, varieties of, 56
Cæcum, diseases of, palpation and percussion in, 475
fæcal impaction of, 476
Calculus, renal, 679
Cancer (*see* Carcinoma).
Capsule internal, lesions of, 800
Carbuncle distinguished from anthrax, 776
Carcinoma, cachexia of, 183
facies of, 118
gastric, supra-clavicular glands in, 134
hemorrhagic exudation of, 167
metastasis in, 183
symptoms of, 182
Cardialgia, 501
Case records, 21
plan for recording, 22
Casts, fibrinous, in plastic bronchitis, 299
Catalepsy, 792
Catarrh, chronic post-nasal, 197
nasal (*see* Rhinitis).
suffocative, 297
Cavities, pulmonary, 263
Cavity, pulmonary, distinguished from pneumothorax, 337
Cerebellum, disease of, 803
Cerebral disease, vomiting in, 500
Chest, adenoid disease of, 236
angles of, 227
auscultation of, ægophony in, 263
bell-tympany in, 262
friction sound in, 261
metallic tinkling in, 261
methods of, 252
pectoriloquy in, 263
position of patient in, 252
pulmonary cavities in, methods of determining, 264
râles in, 259
sounds in disease in, 254
in health in, 253

Chest, auscultation of, stethoscope in, 252
succussion in, 262
vocal resonance in, 262
"barrel-shaped," 232
in adenoid vegetations of naso-pharynx, 458
inspection of, 230
lines of, 227
local changes in size and shape of, 238
mensuration of, 265
movements of, 231
in disease, 238
percussion of, amphoric or metallic sounds in, 251
auscultatory or stethoscopic, 247
cracked-pot sound in, 251
dulness in, 246, 250
methods of, 244
object of, 248
pitch in, 247, 249
pleximeter in, 244
plessor in, 244
position of patient in, 245
resistance in, 247
resonance in, 245, 248
superficial and deep, 247
tympany in, 246, 250
phthisical, 234
rhachitic, 235
regions of, 226
shape and size of, 230
topographical anatomy of, 228
unilateral changes in shape of, 237
Chiasm, optic, diseases of, 807
"Chicken-breast," 187
Chlorosis, 691
venous hum in, 384
Cholera, 760
Asiatic, spirillum of, 150
bacterial diagnosis of, 761
erythema in, 88
facies in, 118
infantum, 550
morbus, 556
distinguished from cholera, 761
nostras, 557
spirillum of, 150
tongue in, 447
Chorea, 845
paralytic, 846
Choreiform movements, 791
Chyluria, 659
parasitic, 660
Circulation, local disturbance of, 172
Cloudy swelling in fever, 106
Coin test in pneumothorax, 336
Colic, 469
hepatic, 526, 615
intestinal, 525
conditions from which it must be distinguished, 526
lead, 526
pancreatic, 502
renal, 526
uterine, 527
Collapse, 116
perspiration in, 76
Colon, dilatation of, 573
in general enlargement of abdomen, 471
Coma, diabetic, 668, 718
uræmic, 667
Comedones, 121
Concussion confounded with shock of hemorrhage, 177
Congestion (*see* Hyperæmia).
Conjunctiva, color of, 124
Constipation, 532
secondary effects of, 533
Constitution, section of, 55
Contractures, hysterical, 851
Convulsions, 792 (*see* Fits).
epileptiform, 791
hysterical, 852
uræmic, 667
Cord (*see* Spinal cord).
Cords, vocal, paralysis of, 816
Corpora quadrigemina, disease of, 801
Corpus callosum, disease of, 799
striatum, 800
Coryza, acute, 194
headache in, 856
syphilitic, 197
Cough, diagnostic significance of, 290
dry, 289
due to enlarged uvula, 452
in aortic aneurism, 424
in carcinoma of œsophagus, 464
in disease of heart, 390
of nose, 189
in ear disease, 289
in laryngeal affections, 204
in mediastinal diseases, 429
in œsophageal affections, 461
in phthisis, cause of, 288
in pleurisy, 333
in pulmonary affections, 288
in tuberculosis, 323
moist, 290
of centric origin, 289
of nervous origin, 205
reflex, 224
stomach, 289
tooth, 289
winter, 298
Coxalgia, distinguished from appendicitis, 571
Cracked-pot sound, 251
Cramp, writer's, 848
Cramps in calves in uræmia, 667
Cranio-tabes, 68, 121
Cranium, fontanelles of, 121
murmur heard over, 381
Crises in locomotor ataxia, 829
Croup, false, 210
membranous, 209
Crus cerebri, disease of, 801
Crystals, 539
Charcot-Leyden, 272, 274
in nasal secretions, 194

Crystals in pus, 167
Cyanosis, causes of, 72
in acute miliary tuberculosis, 318
in congenital heart disease, 419
in emphysema, 304
Cysticercus cellulosæ, 99
Cysts, exploratory puncture of, 160
hydatid, fluid of, 168
ovarian, 169
uric acid in, 170
pancreatic, 170

DECUBITUS, 57
Degeneration, reaction of, 795
Delirium in uræmia, 666
Diabetes insipidus, 719
mellitus, 717
cataract in, 719
complications of, 718
neuralgia in, 855
tongue in, 447
Diaceturia, 648
Diagnosis, ætiological, 18
bacteriological, 145
conditions may render impossible, 19
data upon which based, 18, 24, 49
electrical, 793
general observations on, 17
instruments necessary in, 54
methods of, 19
modern methods of, 20
object of, 18
objective symptoms in, 49
method of observing, 51
requisite knowledge for making, 18
should be complete, 20
temperament and constitution in, 55
value of scars in, 77
Diarrhœa, uræmic, 668
chronic, 531
membranous, 532
microscopical and bacteriological examination of stools in, 530
symptoms of, 530
Diatheses, varieties of, 55
Diphtheria, 755
distinguished from follicular tonsillitis, 456,
laryngeal, 209
Diplococci, 148
Diplococcus pneumoniæ (*see* Micrococcus lanceolatus).
Disease, blue, 419
foot and mouth, 776
Ménière's, 815
mimicry in, 32
present, importance of order of events in, 30
method of eliciting facts in, 29
previous, as a factor in diagnosis, 29
Thomsen's, 137, 832
vagabond's, 74
vertebral, pain in, 46
Diseases, acute infectious, dilatation of heart in, 417
classification of, 23
feigned, 32
infectious, 723
erythemata in, 87
remarks on diagnosis of, 785
roseola in, 87
Dropsy (*see* Œdema).
in nephritis, 669, 672
Drugs, rashes caused by, 86
Duodenum, catarrh of, 549
Dupuytren's contraction, 130
Dysentery, acute, 551
amœbic, 552
diagnosis of, 556
hepatic abscess in, 555
peritonitis in, 556
symptoms of, 552, 555
catarrhal, 551
Dyspepsia, atonic, 519
flatulent, 520
nervous, 519
uterine, 521
Dysphagia in diseases of œsophagus, 463
in laryngeal affections, 204
Dysphonia, 204
Dyspnœa, asthmatic, 285
causes of, 282
clinical varieties of, 287
diagnosis of, 288
due to enlarged uvula, 452
from diminished pulmonary air-space, 284
from muscular inaction, 286
heat, 282
hysterical, 852
in aortic aneurism, 424
in bronchial obstruction, 284
in cardiac disease, 390
in diseases of mediastinum, 429
in nephritis, 672
in pharyngeal affections, 454
in tracheal obstruction, 283
in tuberculosis, 323
laryngeal, 202
distinguished from other forms of, 203
movements in, 125
of emphysema, 288
phrenic, 286
reflex, 224
spasmodic, in interstitial nephritis, 677
uræmic, 667
Dystrophies, connective-tissue, 97

EAR, diseases of, cough in, 289
in general diagnosis, 124
tophi in, 125
Earache in suppurative tonsillitis, 456
Eczema distinguished from varicella, 742
Effusions, specific gravity in, 168
Electrical diagnosis, 793
Elephantiasis, chyluria in, 135

Embolism, 174
cerebral, 841
in arterial sclerosis, 420
kinds of, 175
pulmonary, symptoms of, 175
Embryocardia, 359
in dilatation of heart, 419
Emphysema, 303
adventitious sounds in, 305
atrophic, 305
distinguished from pneumothorax, 336
dyspnœa of, 288
heart in, 305
interlobular, 305
subcutaneous, 97
Empyema, Bacelli's sign in, 331
causes of, 329
in abscess of liver, 611
peptonuria in, 331
points of spontaneous discharge of, 331
pulsating, confounded with aneurism, 428
Endocarditis, chronic, 402
malignant, 400
mycotic aneurism in, 421
simple, 399
Enteralgia, 525, 526
Enteritis distinguished from intestinal obstruction, 566
Entero-colitis, 550
confounded with peritonitis, 577
Enuresis in adenoid vegetations of naso-pharynx, 458
Epiglottis, inflammation of, 210
tuberculosis of, 218
Epilepsy, 849
Epistaxis, causes of, 194
in interstitial nephritis, 676
Eruptions, skin, factitious, 88
traumatic, 89
Erysipelas, 758
Erythema, ætiology of, 84
character of eruption in, 84
classification in, 83
in infectious diseases, 85
kinds of, 84
læve, 85
medicinal, 86
multiforme, 84
nodosum, 86
of infectious diseases, 87
vesicular, 85
Erythromelalgia, 132, 173
Exudations, 161, 167
chylous, 168
serous, 168

FACE, enlargement of, 120
expressions of, 117
hemiatrophy of, 119
in children, 118, 119
in nephritis, 65
in nervous diseases, 119
Face, local affections in skin of, 121
Facial expression in pain, 35
Facies, Hippocratic, 119
in various diseases, 118
Fæces, bacilli of Booker in, 547
bacteria in, 544
blood in, 536
-corpuscles in, 538
chemical examination of, 547
crystals in, 539
epithelium in, 538
fat and pus in, 537
gall-stones in, 536
general section on, 535
microscopical examination of, 537
moulds and yeasts in, 544
mucus in, 537
protozoa in, 539
spirillum of cholera nostras in, 546
tubercle bacillus in, 547
typhoid fever bacillus in, 547
vermes in, 541
Family history in ætiology of disease, 27
Farcy, 773
Fauces (*also see* Pharynx).
examination of, 451
Feet, cold, 133
Fehling's solution, preparation of, 644
Fermentation, putrefactive, 149
Fever (*see* Temperature).
acute specific, 723
"break-bone," 763
cerebro-spinal, 753
clinical causes of, 110
course of, 104
crisis in, 104, 105
definition of, 99
determination of, 100
fastigium in, 104, 111
general causes of, 99
hay, 200, 300
hectic, 178
hemorrhage into skin with, 78
hepatic, 594
high, dangers in, 102
hysterical, 851
in gastric cancer, 508
intermittent, hepatic, 114, 595
irregular forms of, 765
malarial, 763
type of, diseases in which it may occur, 113
urinary, 113
in tuberculosis, 321
lysis in, 104, 105
malarial, 763
miliary, 779
modes of onset in, 105, 111
nervous, 111
pernicious malarial, 769
relapsing, 736
spirillum of, 701
remittent malarial, 768
rheumatic, 705
scarlet, pulse in, 374

Fever, significance of age and sex in, 115
simple continued, 785
symptoms of, 106
syphilitic, distinguished from intermittent, 768
types of, 102, 112
continued, 115
remittent, 114
typhoid, acute bronchitis in, 296
ambulatory, 730
Baruch's diagnostic sign of, 732
bilious, 738
conditions with which confounded, 733
diagnosis of, 732
diazo-reaction of urine in, 727.
distinguished from acute miliary tuberculosis, 318
from influenza, 752
from malignant endocarditis, 401
from pernicious malarial, 770
Eberth's bacillus of, 731
eruption in, 729
facies in, 118
fæcal impaction in, 534
inflammation of parotid in, 461
mode of invasion in, 724
necrosis of sternum in, 139
nervous symptoms in, 727
period of incubation in, 724
pulse in, 374, 727
puncture of spleen in, 161
spleen in, 725
symptoms of, 724
temperature in, 725
varieties of, 730
typhus, 734
cerebro-spinal, distinguished from 736
conjunctivitis in, 124
yellow, 770
Filaria sanguinis hominis, 704
Fingers, abnormal shape of, causes of, 131
tophi in, 130
Fits, 116
Flagellæ, staining of, 154
Flat-foot, pain in, 45
Flatulency in gastric affections, 501
Flushings, significance of, 34
Fontanelles (*see* Cranium).
Food, regurgitation of, in gastric neuroses, 519
Foot, Morton's painful affection of, 45
tabetic, 142
Fremitus, friction, 243
vocal, in disease, 242
in health, 241
Friction, peritoneal, 475
pleural, 261
pleuro-pericardial, 394
Friedreich's sign of cavity, 264
Fungi in saliva, 432

GAIT and attitude in general diagnosis, 57
and station in various nervous affections, 60
Gall-bladder, cancer of, 618
enlargement of, confounded with floating kidney, 617
inflammation of, 616
topographical anatomy of, 597
tumors of, 616
Gall-ducts, gall-stones in, 615
obstruction of, 616
by gall-stones, 618
stenosis of, symptoms, 619
Gall-stones, accidents of, 618
in gall-ducts, 615
Gangrene, 179
of internal organs, 180
Gastralgia, hysterical, 517
in disease of central nervous system, 516
in morphinism, 44
neurasthenic, 517
pain of, 502
Gastrectasia, 512
Gastritis, acute, 504
in onset of various diseases, 505
pain in, 503
vomiting in, 498
chronic, 506
dry mouth in, 430
tabulated diagnosis from gastric ulcer and cancer, 510
vomiting in, 499
mycotic and diphtheritic, 506
phlegmonous, 505
toxic, 505
Gastrodynia, 501
Gastroxynsis, 518
General paralysis of the insane, face in, 119
Gerhardt, "complemental space" of, 228
Gerhardt's sign of cavity, 264
Gingival line, 434
Gland, parotid, inflammation of, 461
Glands, lymphatic, ingeneral diagnosis, 134
Glanders, 773
nasal, 198
Globus hystericus, 202
Glossitis, acute, 439
chronic superficial, 440, 443
dissecting, 441
hemi-, 440
Glycosuria in disease of pancreas, 623
Goitre, exophthalmic, 700
eye in, 123
facies in, 118
pulse in, 374
Stellwag's sign in, 123
Von Graefe's sign in, 123
Gonococcus, 166
Gout, 713
abscesses in, 714
chronic, 714
distinguished from rheumatoid arthritis, 712

Gout, Dupuytren's contraction in, 130
dyspepsia in, 522
joint of, 142
relation to lithæmia, 586
retrocedent, 714
Gram's method, 154
Granulomata, infective, 723
Graves' disease (*see* Goitre).
Gums in lead-poisoning, 434
in scurvy, 434

HÆMATEMESIS, 495
Hæmatidrosis, 721
Hæmatocele, pelvic, 476
Hæmatokrit, 687
Hæmatoma auris, 125
Hæmocytometer, Gowers', 685
Thoma-Zeiss, 686
Hæmoglobinometer, Fleischl's, 684
Gowers', 683
Hæmoglobinuria, 644
paroxysmal, 644
Hæmophilia, 79, 719
conditions with which confounded, 720
degrees of, 720
joint symptoms in, 720
Hæmoptysis, 269
causes of, 291
diagnosis of, 293
distinguished from hæmatemesis, 495
in the invasion of chronic tuberculosis, 319, 323
symptoms of, 292
Hæmothorax, 332
Hair in diagnosis, 120
Hand, claw-, 127, 130
Hands, cold, 133
"Hanging drop," 154
Harrison's groove (*see* Rhachitis).
Hay fever, 300
Headache, "blind," 501
carious teeth, cause of, 435
character of pain in, 859
chronic, 858
congestive, 859
duration of, 859
in fevers, 108
in inflammation of cranial bones, 857
in interstitial nephritis, 675
in lithæmia, 585, 586
in meningitis, 834
in scalp affections, 854
in uræmia, 666
ocular, 855, 859
organic, 859
Hearing impaired by drugs, 125
tests for, 125, 814
Heart, accentuation of aortic second sound of, 357
of pulmonary second sound of, 358
action of, 340
alterations of rhythm of, causes of, 342
anatomy of, 338
aneurism of, 404
Heart, apex beat of, 339, 343, 353
absent, 345
displaced to left, 344
to right, 345
area of deep dulness of, 350
of dulness, method of graphic record, 353
of impaired resonance over, 339
of superficial or absolute dulness of, 348
changes in size of, 349
arrhythmia of, 389
auscultation of, 354
bovine, in aortic insufficiency, 405
cantering rhythm of, 360
chronic valvular disease of, 404
combined valvular lesions of, 414
congenital disease of, 419
dilatation of, 417
definition of, 414
in nephritis, 668
murmurs in, 419
disease of, cerebral symptoms in, 390
cough in, 390
decubitus in, 58
gastric symptoms in, 391
general symptomatology of, 340
inspection in, 343
kidneys in, 391
laryngeal symptoms in, 391
palpation in, 346
percussion in, 348
pleximetric percussion in (*see* Pleximeter).
præcordia in, 343
pulse in, 375
tracings of, 379
retraction of interspaces in, 346
thrills in, 347
tongue in, 448
fatty degeneration of, symptoms of, 403
overgrowth of, symptoms of, 404
feebleness of mitral sound of, 359
of pulmonary sound of, 359
first sound of, accentuated, 357
fœtal rhythm of, 359
hypertrophied, epigastric pulsation in, 369
hypertrophy of, 414
causes of, 414
diagnosis of, 417
in arterial sclerosis, 420
in nephritis, 668
of auricles of, 415
of left ventricle of, 415
of right ventricle of, 415, 416
impulse of, 344, 345
character and strength of, 346
new causes of, 346
in lithæmia, 586
murmurs, causes of, 362
changes in, 366
character of, 365
diagnosis of, 363

Heart murmurs, direction of transmission of, 363, 365
in anæmia, 366
in incompetency of heart valves, 366
in valvular disease, 362
position of maximum intensity of, 363
secondary effect on, and on pulse, of valve lesions, 367
significance of, 366
time of, 364
outline of, on chest wall, 338
pain in disease of, 387
in region of, 385
palpitation of, 388
in interstitial nephritis, 676
reduplicated sounds of, diastolic, 361
systolic, 360
rupture of, 404
second aortic sound of, in arterial sclerosis, 420
sounds of, all increased, 357
all weakened, 357, 359
character of, 356
differentiation of, 357
in health, 354
seat of origin and transmission, 355
systolic and diastolic, differentiation of, 356
valves of, position of, 339
shock of, 347
valvular disease of, dropsy in, 390
dyspnœa in, 390
gastric catarrh in, 522
hemorrhage in, 389
Heartburn (*see* Pyrosis).
Heberden's nodes, 130
Hectic fever, facies in, 118
Heel, pain in, 45
Hemianæsthesia hysterica, 851
Hemianopia, 808
Wernicke's sign of, 808
Hemicrania, 857
Hemiplegia, infantile, 840
nails in, 133
Hemorrhage, 175
cerebral, 837
in aortic aneurism, 424
in cirrhosis of the liver, 605
in hysteria, 851
in leucocythæmia, 694
in splenic leukæmia, 621
internal symptoms of, 176
into skin, 77
meningeal, 822, 839
pancreatic, 623
parenchymatous, 176
Heredity in ætiology of disease, 27
in hæmophilia, 79
in neuroses, 28
in syphilis, 28
method of eliciting facts in, 27
Herpes labialis, 85
Herpes of fifth nerve, 85
pain in, 85
zoster, 85
distinguished from erysipelas, 759
Hiccough in gastric affections, 504
in uræmia, 668
Hodgkin's disease, 696, 697
distinguished from scrofulous glands, 698
Hydrocephalus, 122
chronic, 844
Hydrochloric acid in gastric cancer, 509
Hydronephrosis, 631, 679
Hydrophobia, 777
Hydrothorax, 332
Hyperæmia, 80
causes of, 172
Hyperidrosis, 75
Hyperorexia, 515
Hyperpyrexia, 102
Hysteria, 850
distinguished from meningitis, 836
flatulency in, 501
joint of, 143
Hysterical attacks, 852
Hystero-epilepsy, 853

IMPETIGO distinguished from varicella 742
Indican in intestinal obstruction, 564
in urine, in suppuration, 178
Indigestion in children, in adenoid vegetations of naso-pharynx, 458
intestinal, 547
Infarct, hemorrhagic, 175
of uvula, 452
Inflammation, 177
specific, 723
Influenza, 751
erythema in, 88
Inspection, general section on, 51
systematic, of a patient, 55
Intestinal obstruction, acute, tongue in, 447
confounded with acute hemorrhagic pancreatitis, 624
Intestines, acute catarrh of, 548
amyloid degeneration of, 573
arteries of, emboli of, 525
cancer of, 572
chronic catarrh of, 557
diseases of, 523
constipation in, 532
diarrhœa in, 528
subjective symptoms of, 525
disorders of, in other diseases, 525
hemorrhage of, 534
hernia and volvulus of, course of, 565
infarction of, 573
intussusception of, 564
obstruction of, acute, 559
differential diagnosis in, 563
causes of, 560
urine in, 564

Intestines, obstruction of, chronic, 562
conditions with which confounded, 565
distinguished from peritonitis 565, 577
symptoms of, 561
organic disease of, confounded with colic of, 528
symptoms of worms in, 524
tuberculosis of, 572
ulceration of, 557
general, 558
volvulus of, 559, 565
Intussusception, 476, 559, 564
Iris, actions of, 810
paralysis of, 811
Itching in skin eruptions, 82

JAUNDICE, acute febrile, 595
catarrhal, 614
concomitant symptoms of, 71
fever in, 594
hæmatogenous, 592
distinguished from hepatogenous, 593
hepatogenous, 592
infantile, 593
in tumors of pancreas, 623
malignant, 594
obstructive, diagnosis of cause of, 592
skin in, 71
symptoms of, 591
Joint of synovitis, 141
tabetic, 142
Joints, affections of, distribution in, 144
diagnostically important, 143
enlarged, general causes of, 140
examination of, 144
hysterical, 143

KIDNEY, abscess of, 631, 677
alterations of functions, symptoms, 628
amyloid degeneration of, 678
congestions of, 670
cystic, 170, 678
degenerations of, 677
diseases of, classification, 628
frequency of micturition in, 629
general section on, 627
pain in, 629
enlargements of, causes of, 631
exploratory puncture of, 161
floating, 632, 681
confounded with enlarged gall-bladder, 617
granular or cirrhotic, 674
horseshoe, 679
malignant tumors of, 633
neuralgia of, 629
palpation and percussion of, 630
parasites in, 680
sarcoma and carcinoma of, 678
Kidneys (*also see* Urine).
Koch's laws, 146

LANDRY'S paralysis, 827
Laryngismus stridulus, 213
Laryngitis, acute, 208
with spasm, 210
with stenosis, 209
chronic, 212
phlegmonous, 212
sicca, 209
spasmodic, 210
submucous, 212
Laryngoscopy, 206
Larynx, anæmia of, 208
appearance of, in health, 207
diseases of, 200
conditions confounded with, 201
general symptoms in, 205
sputum in, 207
symptoms of, 201, 205
table of, 211
foreign bodies in, 220
hyperæmia of, 208
in nervous affections, 220
in paralysis of recurrent laryngeal nerve, 215
lepra of, 220
lupus of, 219
muscles of, paralyses of, 214
neuroses of, 213
œdema of, 210
spasm of, 816
syphilis of, 218
tuberculosis of, 216
distinguished from syphilis of, 218
tumors of, 215
Lavage, 488
Lead-poisoning, gums in, 434
Leptomeningitis, cerebral, 834
Leptothrix buccalis, 434
Leucocythæmia, 693
blood in, 694
cover-glass preparations of, 695
staining of, 695
diagnosis of, 696
duration of, 697
splenic, 694
varieties of leucocytes in, 695
Leucocytosis, 688
Leucoderma, 98
Leukæmia, splenic, diagnosis of, 621
Leprosy, 778
bacillus of, 165
Lineæ albicantes, 473
Lipomatosis, 180
Lips in diagnosis, 123
Lipuria, 659
Lithæmia, acute and chronic, 585
headache in, 855
Liver (*also see* Jaundice).
abscess of, 596, 608
confounded with enlarged gall-bladder, 617
empyema in, 611
exploratory puncture in, 611
acute yellow atrophy of, 594
amyloid disease of, 596, 601

Liver, aspiration of, 598
cancer of, 597, 602
cirrhosis of, 604
atrophic, 604
biliary, 607
collateral circulation in, 606
diagnosis of, 607
hemorrhage in, 605
hypertrophic, 603, 607
symptoms and signs of, 605, 606
syphilitic, 607
collateral circulation in portal obstruction, 589
congenital syphilis of, 608
constriction of, from lacing, 597
diseases of, age in, 589
pain in, 45, 590
previous affections in, 590
sex and habits in, 590
stomach in, 522
enlargement of, causes of, 599
pain in, 601
simulated, 599
exploratory puncture of, 160
fatty, 596, 601
floating, 597
functional disturbances of, symptoms of, 584
hydatid disease of, 596, 612
multilocular, 617
hyperæmia of, 587
hypertrophic cirrhosis of, 597
in affections of portal vein, 588
morbid processes in, symptoms of, 584
obstruction of channels of, 587
palpation of, 596
percussion of, 598
pulsation of, in tricuspid insufficiency, 413
symptoms of portal obstruction in, 588
syphilitic gummata in, 608
topographical anatomy of, 590, 598
Locality, sense of, 787
Locomotor ataxia, 828
cephalic crises in, 856
crises in, 43
distinguished from multiple sclerosis, 843
gait in, 60
gastric crises in, 516, 521
joints in, 142
station in, 61
Lumbago, 710
Lung, abscess of, 326
apical consolidation of, subclavian murmur in, 381
collapse of, 326
gangrene of, 325
hydatid disease of, 328
new growths of, 327
Lungs (*also see* Chest).
affections of, reflex vomiting in, 500
and pleuræ, diseases of, cough in, 288
dyspnœa in, 282
hemorrhage in, 291
Lungs and pleuræ, diseases of, pain in, 293
without dyspnœa, 282
capacity of, measurement of, 265
cavities of, 263
congestion of, 306
diseases of, classification of, 222
combination of physical signs obtained in, 266
diagnosis of, 225
friction sound in, 261
inspection and palpation in, 226
palpation in, 241
percussion in, 243
râles in, 259
relation of heart to, 224
of infectious diseases to, 225
respiration in (*see* Breathing).
rhonchi in, 243
subjective and objective symptoms in, 225
symptoms due to external causes in, 223
to morbid processes in, 223
vocal fremitus in, 242
embolism and thrombosis of, 307
gangrene of, distinguished from fœtid bronchitis, 300
hemorrhagic infarcts of, 307
œdema of, 306
topographical anatomy of, 228

MCBURNEY'S point, 568
Macroglossia, 443
Malaria (*also see* Fever).
facies in, 118
hemidrosis in, 76
neuralgia in, 856
plasmodia of, 702
staining of, 703
Malingerer, 32, 37
Marasmus, 62
Measles, 742
distinguished from scarlatina, 748
from varioloid, 741
eruption of, in pharynx, 450
Mediastinum, cancer of, confounded with aortic aneurism, 428
diseases of, 428
Medulla oblongata, lesions of, 803
Melanæmia, 688
Membranes, inflammation of, 179
Ménière's disease, 61, 815
Meninges, tumors of, 842
Meningitis, chronic, 836
cerebro-spinal, distinguished from influenza, 752
distinguished from typhoid fever, 733
purulent, 836
spinal, 820
chronic, 821
syphilitic, 821, 836
tubercular, 834
conjunctivitis in, 124

Meningitis, tubercular, distinguished from simple form, 835
Metallic tinkling, 336
Micrococci, 148
Micrococcus lanceolatus, 162, 166
in pleural effusion, 168
in sputum, 280
staining of, 280
Microcythæmia, 688
Micro-organism (*see* Bacteria *or* Bacteriology).
Microscope, diaphragm of, use of, 152
Micturition, frequent, causes of, 629
Migraine, 857
Miliaria, 88
Milk sickness, 779
Mitral incompetency, 407
bloodvessels in, 410
broken compensation in, symptoms of, 409
diagnosis of, 410
effects of, upon the circulation, 408
other valvular murmurs in, 410
physical signs of, 409
thrill in, 348
stenosis, 410
associate murmurs in, 412
physical signs of, 411
presystolic thrill in, 347
Morbid processes, 171
in tubes or channels, 184
symptomatology of, 171
Morphinism, pain in, 36, 44
Motility, disturbance of, 790
Mouth, diseases of, 430
dry, 430
eruptions in, in infectious diseases, 431
inflammation of (*see* Stomatitis).
secretions of (*see* Saliva).
ulcers of, herpetic, 438
syphilitic, 438
Mumps, 753
face in, 120
Murmur (*see* Heart).
in disease of arteries, 382
functional or hæmic, 381
pressure, 381
respiratory, 253
Muscles, atrophy of, 135
primary, 136
Raymond's table of, 138
hypertrophy of, 137
lack of tone in, 115
ocular, spasm of, 813
progressive ossification of, 138
pseudo-hypertrophy of, 135
Myelitis, 823
acute transverse, 824
disseminated, 824
central, 825
chronic, 825
Myocarditis, 402
Myositis, 138
Myotonia congenita, 137
Myxœdema, 96
dry skin in, 77
outline of face in, 66

NAILS, shape and color of, 132
trophic changes in, 133
Naso-pharynx, adenoid vegetations of, 457
affections of, in children, 450
Nausea in gastric affections, 496
Neck in general diagnosis, 125
Necrosis, 179
Nephritis (*also see* Uræmia).
acute exudative, 671
with excessive pus, 672
occurring with tonsillitis, 455
productive or diffused, 672
anæmia in, 670
cardio-vascular symptoms of, 668
chronic productive, with exudation, 673
course of, 674
without exudation, 674, 676
apoplexy in, 676
gastro-intestinal symptoms in, 670
heart in, 675
inflammation of serous membranes in, 676
neuro-retinitis in, 676
pulmonary symptoms in, 675
uræmia in, 675
urine in, 674
classification of, 671
dropsy in, 669
face in, 669
gastric symptoms in, 522
hemorrhages in, 669
in scarlatina, 747
interstitial, 674
ophthalmoscopic changes in, 669
petechiæ in, 670
suppurative, 677
tubercular, 677
Nephrolithiasis, 679
Nerve, recurrent laryngeal, paralysis of, 215
Nerves, diseases of auditory, 814
of fifth, 811
of fourth, 811
of glosso-pharyngeal, 815
of hypoglossal, 817
of laryngeal, 815
of oculo-motor, 810
of olfactory, 806
of optic, 806
of pneumogastric, 815
of seventh, 813
of sixth, 812
of spinal accessory, 817
Neuralgia, 789
carious teeth, cause of, 435
cephalic, 855

Neuralgia, intercostal, 385
distinguished from pleurisy, 334
lumbo-abdominal, confounded with intestinal colic, 528
of kidney, 629
of nerves of scalp, 855
of spinal nerves, 818
trifacial, 812
Neurasthenia, 853
Neuritis confounded with joint affections, 141
multiple, 819
nails in, 133
of brachial plexus, 818
of spinal nerves, 817
optic, 807
peripheral, in diabetes mellitus, 718
wrist-drop in, 128
Nigrities, 439
Nodules, subcutaneous, 99
Nose, auxiliary cavities of, disease of, 199
in adenoid vegetations of naso-pharynx, 457
deformity of, 190
disease of (*see also* Rhinitis).
asthma in, 200
color of mucous membrane in, 192
rhinoscopy in, 191
symptoms of, 187
ulceration of mucous membrane in, 193
ulcerative, 199
foreign bodies in, 198
polypi of, 198
tumors of, 198
Nystagmus, 811

OBESITY in general enlargement of abdomen, 470
Occupation in ætiology of disease, 25
Œdema, angio-neurotic, 95
anthrax, 775
causes of, 92
combined, 96
conditions with which confounded, 93
definition of, 92
general, 95
local, 94
of arms and thorax, 94
of face, 94
of feet, 94
of larynx, 210
in angio-neurotic œdema, 96
recognition of, 93
sudden, of thorax, 95
Œsophagus, abscess of, 464
acute inflammation of, 464
affections of, 461
dysphagia in, 463
functional, 465
subcutaneous emphysema in, 462
carcinoma of, 464
chronic inflammation of, 464
dilatation of, 465
Œsophagus, foreign bodies in, 465
hemorrhage from, 462
normal constriction of, 462
paralysis of, 466
physical examination of, 462
stricture of, 464
Oligæmia, 172
Oligocythæmia, 688
Ophthalmoplegia, 813
Osteitis deformans, 66
Osteomalacia, 68
Osteomyelitis, 140
Ovary, cysts of, fluid of, 169
tumors of, 477
Ozæna, 197
in glanders, 774

PACHYMENINGITIS, cerebral, 833
cervical hypertrophic, 821
spinal, distinguished from myelitis, 826
Pain, abdominal, character of, 469
due to disease of abdominal walls, 469
vertebral disease, 469
in uræmia, 667
and tenderness in stomach, 485
character of, 40
in inflammation of various tissues, 179
chest, in tuberculosis, 323
chronic, in back, 48
clinical value of, 38
crises of, 43
definition of, 35
duration of, 39
epigastric, 386
causes of, 501
estimation of degree of, 38
facial expression in, 35
gastric, 527 (*see* Gastralgia).
in abscess of liver, 609
in affections of intestines, 525
in aortic aneurism, 423
in appendicitis, 568
confounded with intestinal colic, 527
in carcinoma, 182
in cardiac region, causes of, 385
in diaphragmatic pleurisy, 333
in diseases of kidneys, 629
of lungs and pleuræ, 293
of mouth, 430
in ear, in tonsillitis, 456
in enlargement of liver, 601
in extremities, 44
forerunner of apoplexy, 45
in gastric affections, 45, 502
ulcer, 485
in girdle sensation, 46
in head, 854
in hepatic disease, 590
in herpes, 85
in joint affections, 141
in local peritonitis, 527

Pain in loins, 47
in Morton's painful affection of the foot, 45
in neuritis of spinal nerves, 818
in œsophageal affections, 461
in pharyngeal affections, 454
in pleurisy, 333
in rectal ulcer, 42
in side, 47
in spine, 46
in toxæmias, 41
in vertebral disease, 46
location of, 41
methods of recognizing objectively, 35
modes of onset of, 39
modified by pressure, movement, rest, 43
pancreatic, 527
pathology of, 35
peripheral, of central origin, 42
posture in, 36
radiating, 42
rectal, 527
reflex actions due to, 36
in gastric disease, 45
in liver disease, 45
in thorax, 45
sensation of, 788
simulated, 37
sources of error in estimating, 36
sympathetic or reflex, definition of, 35
time of occurrence, 40
variations of, in disease, 35
Palpation, general section on, 52
Palsies, local, in uræmia, 667
Pancreas, affections of, pain in, 502
cyst of, 170, 625
diseases of, 622
hemorrhage into, 623
tumors of, 478, 623
Pancreatitis, acute hemorrhagic, 624
gangrenous, 625
hemorrhagic, distinguished from intestinal obstruction, 566
suppurative, 624
Paræsthesia, 34
Paralbumin in ovarian fluid, 169
tests for, 170
Paralysis, 790
acute ascending, 827
agitans, 846
distinguished from multiple sclerosis, 844
gait in, 60
alternating, 801
Bell's, 813
bulbar, larynx in, 220
diver's, 827
general, of insane, distinguished from multiple sclerosis, 843
glosso-labial, face in, 119
-laryngeal, 844
hysterical, 851
of fifth nerve, 812
peripheral facial, face in, 119
Paralysis, pseudo-hypertrophic muscular 831
gait and station in, 61
vasomotor, 793
Paramyoclonus multiplex, 137
Paraplegia, ataxic, 830
hysterical, distinguished from myelitis, 826
gait in, 60
primary spastic, 829
spastic, cross-legged progression in, 61
gait in, 60
Parasites, 147
Parotitis, 461
Pectoriloquy, 263
Peliosis rheumatica, 722
Peptonuria in empyema, 331
Percussion (*see* Chest).
general section on, 53
Pericarditis, acute fibrinous, 393
causes of, 392
friction in, 348
mediastinal, 399
with effusion, 394
diagnosis of, Bamberger's sign in, 397
physical signs of, 395
pressure symptoms in, 394
Pericardium, adherent, 398
Friedreich's sign in, 398
pulsus paradoxus in, 398
air in, 398
blood in, 398
diseases of, pain in, 386
friction sound of, 361
distinguished from pleural friction, 362
from pleuro-pericardial friction, 362
points for puncture of, 160
serum in, 397
Perinephritis, 632
Peristalsis, unrest of, in gastric neuroses, 519
Peritoneum, cancer of, 580
tuberculosis of, 580
Peritonitis, 574
abdominal walls in, 475
acute tuberculous, confounded with appendicitis, 571
tuberculous, swelling in pubic region in, 478
chronic, 578
conditions with which confounded, 577
confounded with intestinal colic, 528
diagnosis of, 576
distinguished from intestinal obstruction, 565
friction sound in, 475
hysterical, 577
local circumscribed, 577
pain in, 527
symptoms of, 575
vomiting in, 500
Perspiration, diminished, 77

Perspiration, excessive, conditions in which it may occur, 76
local, 76
prolonged, 76
Pertussis, 750
Petri's plates, 156
Pharyngitis, acute, 459
chronic, 460
granular, 460
phlegmonous, 459
rheumatic, 460
spasm of pharynx in, 454
Pharynx, affections of, cervical glands in, 453
subjective symptoms of, 454
anæsthesia of, 453
and fauces, diseases of, 449
eruption of measles in, 450
eruptions in, 451
exudations on, 453
hyperæsthesia of, 453
paralysis of, 450
spasm of, 454
ulceration of, cancerous, 453
follicular, 452
syphilitic, 452
tuberculous, 453
Phosphates, triple, in pus, 167
Phthisis (*see* Tuberculosis).
confounded with aortic aneurism, 423, 428
Plague, the, 777
Plethora, 172
Pleura, air in, distinguished from emphysema, 267
aspiration of, 160
effusions into, decubitus in, 58
pulsating effusion of, 332
thickened, 332
Pleuræ, affections of, 222
air in lungs in, 224
effusion into, confounded with enlarged liver, 599
distinguished from pulmonary consolidation, 267
movement of chest in, 240
friction sound of, 261
Pleurisy, acute, 329
ægophony in, 330
bronchial breathing in, 257, 330
causes of, 329
chronic, 334
diagnosis of, 333, 334
diaphragmatic, 333
displacement of organs in, 330
effusion in, physical signs of, 330
exploratory puncture in, 333
pain in, 293
conditions with which confounded, 294
purulent (*see* Empyema).
rheumatic, 707
Skoda's resonance in, 330
tuberculous, 332
Pleurodynia, 385, 710
Pleurodynia, distinguished from pleurisy, 240, 334
Plexímeter, Sansom's, 351
data obtained by, 352
repercussion with, 352
Pneumococcus (*see* Micrococcus lanceolatus).
Pneumonia, broncho-, distinguished from collapse of the lung, 327
catarrhal, 313
diagnosis of, 314
physical signs of, 313
tuberculous form of, 314
chronic interstitial, 314
croupous or lobar, 308
bacteriological diagnosis in, 313
bilious, 312
central, 308
cerebral symptoms in, 310
complications in, 313
cutaneous symptoms in, 310
diagnosis of, 312
distinguished from collapse of the lung, 327
duration and course of, 311
fever in, 310
gastro-intestinal symptoms in, 310
heart and pulse in, 310
in wasting diseases, 312
micrococcus lanceolatus in (*see* Sputum).
migratory, 312
mode of onset of, 308
physical signs of, 311
pseudo-crisis in, 310
pulse-respiration ratio in, 309
respiratory symptoms in, 308
rusty sputum of, 309
typhoid, 312
urine in, 311
distinguished from influenza, 752
from pleurisy, 334
facies in, 118
movement of chest in, 240
sputum in, 269
without bronchial breathing, 257
Pneumonokoniosis, 314, 315
Pneumoptosi in gastric neuroses, 518
Pneumothorax, 335
diagnosis of, 336
movement of chest in, 240
signs of, 336
Poikilocytosis, 688
Poliomyelitis, anterior, 826
chronic, 831
Pons, lesions of, 801
Posture in diagnosis, 57
in pain, 36
Pregnancy, extra-uterine, cause of pelvic hæmatocele, 477
confounded with uterine colic, 527
Pressure, sense of, 788
Proctitis, 550
Pruritus in uræmia, 667
Ptomaïnes, 149

Ptosis, 811
morning, 811
Ptyalism, mercurial, 437
Puerperium, pulse in, 375
Pulmonary insufficiency, 414
osteo-arthropathy, 66
stenosis, 414
Pulsation, epigastric, 368, 484
of organs, 370
Pulse affected by condition of arterial walls, 372
capillary, in aortic incompetency, 406
Corrigan's or water-hammer, 367
frequency of, 370
diagnostic significance of, 374
in aortic aneurism, 426
in dilatation of heart, 418
in hypertrophy of left ventricle, 416
intermittent, 373
irregular, 374
method of taking, 371
rate of, in fever, 106
rhythm of, 373
sphygmographic tracings of, interpretation of, 378
tension of, high, causes of, 372
in true and false angina pectoris. 373
low, 373
venous, 383
volume of, 373
Pupil, Argyll-Robertson, 811, 829
Purpura, 721
Pus, 161
abnormal, 167
amœba dysenterica in, 162, 166
chemical examination of, 167
crystals in, 167
protozoa in, 166
sero-, 167
vermes in, 167
Pyelitis, 680
Pyonephrosis, 631, 680
Pyo-pneumothorax subphrenicus, 337, 479
Pyrosis in gastric affections, 503, 518

QUINSY, 456

RALES, crepitant, in influenza, 752
kinds of, 259
recognition of, 260
Ranula, 440, 443
Rashes caused by drugs, 86
Raynaud's disease, 131
Recording cases (*see* Case records).
Rectum, diseases of, 574
Reflexes, cutaneous, 792
delay in, 792
patellar, 793
in diphtheria, 756, 758
tendon, 793
Resonance, vocal, 262
Skoda's, 330
Respiration, Cheyne-Stokes, 239
cause of, 285
in fever, 106
Retinitis, 806
albuminuric, 806
Rhachitis, 67, 715
face in, 119
laryngismus in, 213
perspiration of head in, 77
Rheumatic diathesis, relation of stomach to, 522
Rheumatism, abdominal, confounded with intestinal colic, 528
acute articular, 705
complications and sequelæ of, 707
diagnosis of, 708
duration of, 708
endocarditis and pericarditis in, 707
subcutaneous nodosities in, 708
affections of tonsils in, 450
chronic articular, 709
gonorrhœal, joint of, 141
joint of, 142
muscular, 710
nodules in, 99
of abdominal walls, confounded with peritonitis, 577
pain in, 469
relation to lithæmia, 586
subacute articular, 709
Rheumatoid arthritis (*also see* Arthritis).
freckles in, 75
Gubler's tumor in, 129
joint of, 142
outline of joints in, 140
Rhinitis (*see* also Nose).
atrophic, 197
caseous, 193
chronic hypertrophic, 196
diphtheritic, 195
secretions in, 193
sicca, 197
simple acute, 194
Rhinoliths, 198
Rhinoscopy, 191
Rhinorrhœa, idiopathic, 200
strumous, 197
Rhonchi, 243, 259
Rhythmical contractions, 791
Ribs, periostitis of, in præcordial region, 385
Roseola, differential diagnosis from acute infectious diseases, 87
Rubella, 749
distinguished from scarlatina, 748

SALIVA, chemical examination of, 432
fungus of thrush in, 433
in disease, 433
micro-organisms in, 432
microscopical examination of, 431

Saliva, sugar in, test for, 433
sulpho-cyanide of potassium in, 433
Salivation, 437
Salpingitis confounded with typhoid fever, 733
Sapræmia, 147
Saprophytes, 147
Sarcinæ, 148
Scalp, affections of, pain in, 854
Scarlatina, 744
anginosa, 747
Scars, significance in diagnosis, 77
Schizomycetes, 147
Sciatica, 819
in tumor of kidney, 678
Scleroderma, 98
Sclerosis, disseminated insular, gait in, 60
lateral, 827
multiple, 843
speech in, 220
posterior, larynx in, 220
Scurvy, 716
brawny induration in, 98
gums in, 434
hemorrhage in, 79
Seitz's sign of cavity, 264
Sensation, delayed conduction of, 788
Sense, muscular, 788
Sensibility, disturbances of, 787
tactile, 787
Sex in ætiology of disease, 25
Shock, 117
causes of, 177
Sinus, frontal, inflammation of, 189, 199
sphenoidal and ethmoidal, inflammation of, 199
Skin, bronzed, 73
conditions with which confounded, 74
color of, 68
in various diseases, 70
deep affections of, 91
eruptions of, in internal diseases, 79
anatomical characters of, 80
associated morbid phenomena in, 81
clinical significance of, 80
distribution of, 81
general health in, 82
itching in, 82
table of, with diseases in which they may occur, 83
glossy, 132
hemorrhage into, 77
cause of, 78
in fever, 78
significance of, 78
Vierordt's test for, 78
inflammations of, superficial, 91
moisture and dryness of, 75
nutrition of, 75
scars of, 77
Skoda's resonance, 330
"Sleeping sickness," 704
Smell, sense of, disturbance of, 189
Solutions, Gabbett's, 279
Gibbes', 279
Koch-Ehrlich, 278
of aniline dyes, 153
Ziehl-Neelsen, 278
Spanæmia, 172
Spasms, 791
coördinated, 792
tonic, 792
vasomotor, 793
Speech, centres of, 797
Spermatorrhœa, 657
Sphygmograph, 375
Dudgeon's, directions for using, 376
technique of, 377
Spinal column, curvature of, 139
cord, anæmia of, 823
anterior horns of, 804
antero-lateral columns of, 804
compression of, 823
degenerations of, 828
hemorrhage into, 822, 827
hyperæmia of, 823
localization of, 803
posterior columns of, 804
tumors of, 833
unilateral lesions of, 804
Spine, pain in, 46
Spirilla, 150
of Finkler and Prior, 150
Spirillum choleræ Asiaticæ, 545
cholera nostras, 546
Obermeieri, 737
Spirometry, 265
Spleen, acute enlargement of, 620
distinguished from enlarged kidney, 620
amyloid, 621
chronic enlargement of, 621
enlargement of, in young children, 622
exploratory puncture of, 160
floating, 619, 620
hydatid tumor of, 622
in chronic malaria, 621
in cirrhosis of liver, 605
in dilatation of stomach, 513
malignant tumors of, 622
palpation of, 619
percussion of, 620
syphilis of, 622
Splenitis, acute, 620
Spores of bacilli, 149
staining of, 154
Sputum, actinomyces in, 281
alveolar epithelium in, 270
bacillus of influenza in, 281
chemistry of, 281
collection of, for examination, 152
crystals in, 274
elastic fibres in, 271
examination of, 268
fibrinous coagula in, 272
in amœbic hepato pulmonary abscess, 556
in œsophageal affections, 461

Sputum in bronchiectasis, 300
laryngeal, 207
method of collecting, 267
micrococcus lanceolatus in, 280
parasites in, 275
spirals in, 272
tubercle bacillus in, 276
detection of, if few in number, 279
importance of, 279
methods of staining, 277
varieties of, 268
Staining, of blood in leucocythæmia, 695
methods of, 153
Staphylococci, 148
pyogenic, 162
Station (*see* Gait).
Sterilization in bacteriology, 151
intermittent, 151
Stethoscopes, 252
Stomach, absorptive energy of, test for, 493
actual diminution in size of, 486
artificial distention of, by gas or air, 486
atony of, 519
and atrophy of, 507
auscultation of, 487
bougie, its use and contra-indications in examination of, 484
cancer of, 484, 507
diagnosis of, 509
examination of gastric contents in, 509
pain in, 502
tabulated diagnosis from gastric ulcer and chronic gastritis, 510
vomiting in, 499
contents of, alcohol in, 493
and activity of digestion in disease of, 482
as index of digestive energy, 490
bile and intestinal juice in, 489
blood in, 489
carbohydrates in, test for, 493
fatty acids in, 492
HCl in, quantitative estimation of, 491
significance of, 492
tests for, 490
inspection of, 489
lactic acid in, 492
methods of securing, for examination, 487
microscopical examination of, 494
mucus in, 489
pepsin in, test for, 493
peptones in, test for, 493
pus in, 490
rennet in, test for, 493
test breakfast in examining, 490
total acidity of, 492
digestive energy of, Günsburg's test for, 493
dilatation of, 486, 512
diseases of, 480
Stomach, diseases of, alterations of appetite in, 503
central and reflex influences in, 481
constipation or diarrhœa in, 504
flatulency in, 501
functional symptoms of, 481
hiccough in, 504
morbid processes in other organs causing symptoms of, 483
organs functionally related, 483
pain in, 45, 46, 501
pyrosis or water-brash in, 503
regurgitation of gas or food in, 503
subjective symptoms in, 496
symptomatology of, 480
toxic symptoms in, 483
vertigo in, 501
vomiting in, 495
hemorrhage from, 495
causes of, 495
hyperacidity and hypersecretion of, in gastric neuroses, 517
local diseases of, vomiting in, 498
method of passing tube into, 488
motor power of, test for, 494
neuroses of, 513 (*also see* Dyspepsia).
diagnosis of, 520
Ewald's table of, 513
non-malignant tumors of, 484
normal contents of, characters of, 488
pain and tenderness in, 485
palpation of, 484
percussion area of, 486
peristaltic and anti-peristaltic waves of, 483
physical examination of, 483
position of, 485
rupture of, 513
succussion splash of, 487
symptoms of affections of, in other diseases, 521
tympany of, distinguished from that of colon, 486
ulcer of, 510
pain in, 502
tabulated diagnosis from gastric cancer and chronic gastritis, 510
vomiting in, 499
Stomatitis, aphthous, 436
catarrhal, 436
gangrenous, 437
materna, 437
mercurial, 437
parasitic, 437
ulcerative, 436
Streptococci, 148
pyogenic, 163
Succussion, Hippocratic, 262, 336
Sudamina, 88
Suppuration, fever of, 178
indican in urine in, 331
loss of appetite attending, 503
peptonuria in, 331
Suppurations, contagious, 723

Sweating sickness, 76, 779
Sweats in tuberculosis, 323
Symptoms, cerebral, in fever, 107
of disease, objective, definition of, 17
in general diagnosis, 49
subjective, definition of, 17
general, 33
nature of, 33
value of, 31
Syphilis, brawny induration in, 98
caries of frontal bone in, 121
congenital, eruptions in, 90
teeth of, 435
constitutional, 780
eruptions of, 89
hereditary, 782
neuralgia in, 856
tenderness of sternum in, 139
tertiary ulcers of, 90
ulcers of pharynx in, 452
Syringomyelia, 833

TABES dorsalis (*see* Locomotor ataxia).
mesenterica, 480
Tachycardia, 374
Tæniæ, 541
symptoms of, 524
Teeth, carious, cause of headache or neuralgia, 435
dates of eruption of, 436
effect of stomatitis on, 435
in hereditary syphilis, 782
of congenital syphilis, 435
Teething, 435
Temperament, section on, 55
Temperature (*also see* Fever).
collapse, 102
determination of, 100
febrile, 102
hectic, 76
hyperpyretic, 102
in uræmia, 675
in uræmic convulsions, 667
inverted, 105
recrudescent, 105
sense of, 788
subnormal, 109, 112
sudden fall in, 112
rises in, 111
variations of, pathological, 102
physiological, 101
Tetanus, 847
bacillus of, 166
Tetany, 847
Thalamus, optic, 800
Thermometer, 100
Thirst as a symptom, 496
Thorax (*see* Chest).
reflex pains in, 45
Thrombosis, 173
causes of, 174
cerebral, 841
in arterial sclerosis, 420
inflammatory or septic, 174
Thrombosis, of superior longitudinal sinus, 840
Thrush, 433, 437
Thyroid, enlargements of, 126
Tic douloureux, 789, 812
Tinea versicolor confounded with Addison's disease, 74
Tongue, atrophy of, 443
black, 439
chancre of, 442
coating on, 444
cyanosis of, 445
cysts of, 443
dry brown, 445
effects of food on, 447
eruptions on, 440
examination of, 438
furred or shaggy, 445
furrows of, 440
hypertrophy of, 443
indentations and excoriations of, 440
inflammation of (*see* Glossitis).
in angina Ludovici, 449
in relation to diseases of the alimentary canal, 447
in treatment and prognosis, 448
in various diseases, 448
moisture of, 447
movements of, 448
mucous patches on, 443
patches and plaques on, 442
plaster, 445
red dry, 445
sclerosis of, 441
stippled or dotted, 444
strawberry, 444, 746
syphilitic fissures of, 441
tuberculous ulcer of, 442
ulcers of, 441
wandering rash on, 443
xanthelasma of, 439
Tongues, classification of, 446
Tonsillitis, catarrhal or erythematous, 454
chronic, 457
diagnosis of, 456
follicular, 455
distinguished from diphtheria, 456, 757
odor of breath in, 454
herpetic, 455, 456
suppurative, 456
Tonsils, 451
affections of, in rheumatic states, 450
exudations on, 453
foreign bodies in, 457
leptothrix of, 453
Torticollis, 710, 817
Toxæmia, 147
Toxalbumins, 149
Tracheal tugging, detection of, 126, 427
Tract, optic, diseases of, 807
Transudations, specific gravity in, 168
Tremor, 791
Triangle, Simon's, 739

Trichina spiralis in fæces, 543
Trichinosis, 779
face in, 120
œdema in, 95
Tricuspid incompetency, 412
venous pulse in, 384
stenosis, 413
Trophic disturbance, 793
Tubercle bacillus in pus, 163
Tuberculosis, 783
acute miliary, 317, 784
pulmonary, distinguished from typhoid fever, 733
chronic, 318
diagnosis of, 325
diagnostic features of, 321
early signs and seats of, 319
infection in, 324
modes of invasion of, 318
physical signs of, 321, 324
progress of, 320
sputum in, 324
vomiting in early stages of, 324
dyspeptic symptoms in, 521
hæmoptysis in, 291
intermittent type of fever in, 113
of kidney, 677
of larynx, 216
of mesenteric glands, 480
of occipito-atlantal articulation, 121
pulmonary, acute, 315
definition of, 315
distinguished from pneumonia, 317
expectoration in, 317
physical signs of, 316
pneumothorax in, 335
Tubes, Esmarch's, 156
Tumors, of abdomen, 472
phantom, 472
Tympanites hysterical, 852
Typhlitis, 571
tumor of, 476
Typhoid fever (*see* Fever).
state described, 107
in nervous affections, 107

ULCER, duodenal, 557
gastric, pain and tenderness in, 485
Uræmia, cerebral symptoms of, 666
chronic, confounded with typhoid, 734
distinguished from apoplexy, 839
dyspnœa in, 667
gastro-intestinal, symptoms of, 667
in chronic productive nephritis, 673, 675
vomiting in, 500
Urine, acetone in, 648
albumin in, 639
boiling test for, 639
necessity of clear solution in testing for, 641
Urine, albumin in, nitric acid test for, 640
picric acid test for, 640
quantitative estimation of, 641
albumose in, 644
bile pigments and bile acids in, 647
blood in, 643, 649
calcium oxalate in, 664
casts in, method of examining for, 652
varieties of, 655
chlorides in, 639
cholesterin in, 665
color of, normal and abnormal, 633
cylindroids in, 656
cystin in, 665
density of, 636
diacetic acid in, 648
diazo-reaction of, in typhoid, 727
entozoa in, 660
epithelium in, 659
extraneous matters in sediments of, 649
fat in, 659
hæmoglobin in, 644
indican in, 647
in acute exudative nephritis, 671
productive nephritis, 672
in amyloid disease of kidney, 678
in chronic productive nephritis, 673, 674
in lithæmia, 586
in organic heart disease, 391
in pyelitis, 680
in tumors of kidney, 661
leucin and tyrosin in, 665
melanin in, 666
microscopic examination of, 648
micro-organisms in, 660
mucin in, 643
odor of, 637
peptone in, 643
phosphates in, 663
preservation of specimens of, 650
pus in, 648, 651
reaction of, 636
sediments in, 637
centrifugal machine for securing, 649
solids in, specific gravity as index of, 636
spermatozoa in, 657
sugar in, 644
Fehling's test for, 644
fermentation test for, 645
phenyl-hydrazin test for, 645
quantitative estimation of, 645
suppression of, causes of, 635
urates in, 663
urea in, 638
uric acid in, 662
murexid test for, 637
volume of, in disease, 635
Urticaria, 86
Uvula, affections of, 452

VALVE, ileo-cæcal, stricture of, 477
Varicella, 741
distinguished from varioloid, 741
Variola, 738, 740
Varioloid, 740
Vasomotor apparatus, 186
Vein, portal, suppurative inflammation of, 588
symptoms of obstruction of, 588
Veins, hum heard over, 384
increase in size of, causes of, 382
pulsation of, 383
in tricuspid insufficiency, 413
pulse of, 383
rhythm of, 383
thrombosis of, 384
Vermes, round, 542
tæniæ, 541
trematodes or flukes, 542
Vertigo, aural, 815
in gastric affections, 501
paralyzing, 815
Volvulus, 559
Vomiting, 496
determination of, cause of, 498
in Addison's disease, 699
in chronic gastritis, 506
Vomiting in intestinal obstruction, 561
in local affections of stomach, 498
in onset of acute diseases, 499
in toxæmias, 500
nervous, in gastric neuroses, 519
physiology of, 497
reflex, 499
uræmic, 667
Von Graefe's sign, 701

WASTING in aortic aneurism, 423
Weight in relation to height, 63
Hutchinson's table of, 64
Weil's disease, 595
Whooping-cough, 750
sublingual ulcer in, 438
Wintrich's signs of cavity, 264, 336
Worms, intestinal, symptoms of, 524
Wrist-drop, 128

XANTHELASMA, 123

ZOÖGLŒÆ, 148

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